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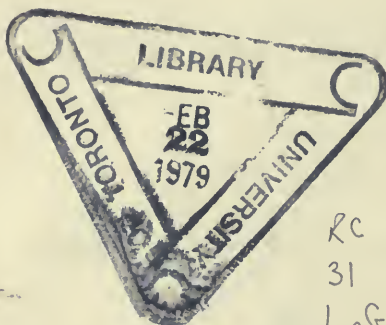
GUY'S HOSPITAL REPORTS.

EDITED BY
H. G. HOWSE, M.S.,
AND
FREDERICK TAYLOR, M.D.

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 Morris, Henry, M.A., M.B., 2, Mansfield Street, Portland Place, W.
 Morris, John, 30, Dorset Gardens, Brighton
 Morse, R. E. R., Eton House, Oriel Terrace, Cheltenham
 Morse, T. H., 12, Prince of Wales's Road, Norwich
 Moxon, H. J., Guy's Hospital
 Moxon, Walter, M.D., 6, Finsbury Circus, E.C.
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 Murphy, S. F., 158, Camden Road, N.W.

 Nason, John James, M.B., Stratford-on-Avon
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 Owen-Jones, Percy, 662, W. Van Buren Street, Chicago, Illinois,
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Le Progrès Médical (per Dr. Bourneville, Rue des Écoles 6, Paris).
Chicago Medical Journal and Examiner (per Dr. W. H. Byford, care of Messrs. W. B. Keen, Cooke, & Co., 113 and 115, State Street, Chicago, United States of America).
Proceedings of the Medical Society for the County of Kings (Society Rooms, Everett Hall, 398, Fulton Street, Brooklyn, New York, U. S. America).
Transactions of the American Medical Association (per Messrs. Churchill).
Transactions of the American Gynæcological Society, Clarendon Street, Boston, Mass. (per Messrs. Churchill).
Library of Surgeon-General's Office, U.S. Army, Washington, D.C. (per Mr. B. F. Stevens, U.S. Government Despatch Agency, 4, Trafalgar Square, London, W.C.).
Transactions of the Brooklyn Anatomical and Surgical Society, 28, Madison Street, Brooklyn, New York, U.S.A.
The Sanitary Record, 15, Waterloo Place, S.W.
Mémoires de la Société de Médecine et de chirurgie de Bordeaux (per Dr. Demons, Hôpital St. André, Bordeaux).

In Memoriam.

JOSEPH TOWNE,

MODELLER TO GUY'S HOSPITAL FOR FIFTY-THREE YEARS.

By THOMAS BRYANT.

INSTITUTIONS cannot afford, more than nations, either to forget their great men, or to fail to honour, however inadequately, the mental qualities or doings of those who, by the possession of great, or the skilled use of average abilities, tower in one form or another above the level of the many, amidst whom they labour. And it may be added, that the biography of great men is not the least instructive or interesting portion of a nation's literature. With this feeling I write this short biography of my old friend Joseph Towne, the late well-known, highly esteemed, skilled modeller of Guy's Hospital, who, as a youth of seventeen years of age, became attached to the institution in the year 1826; who worked for it for upwards of fifty years, and died, full of honour and deeply regretted, in harness, on June 25th, 1879.

From a letter before me, written by one of his daughters, I find he was the third son of Mr. Thomas Towne, a dissenting minister of the country village of Royston, near Cambridge, and that he was born on November 25th, 1808. It is also said that his

education, even for the period, was very slender, and that his life, till his connection with Guy's, was passed in a quiet country home.

As a child, his great amusement was modelling in clay, and one of his daughters has now in her possession a miniature model of a monkey, which represents an early expression of talent. Till sixteen years of age he modelled for amusement in a desultory way, but beyond the existence of the monkey, it is believed that no other early specimens of his work are now extant.

His father, however, during these years, was not unobservant of what he saw in his son, and being himself possessed of some artistic talent as a painter, he felt the wisdom of encouraging and aiding his son's natural bent. Under these circumstances he entertained the idea of making him a pupil of the distinguished sculptor, Behnes; and if the son had not been keener than his father as to his future career, it is probable that his talent would have been diverted into another channel, and expended in a groove, which would not have been that of a modeller.

It so happened, however, that Towne's first work of importance was the model of a human skeleton, thirty-three inches high, which, through the kindness of his family, now stands in a place of honour in the museum of Guy's, on a bracket which overhangs his own wonderful models; and when it is known that this model was made when Towne was but seventeen years of age, that it was moulded in a certain sense secretly, and built up from drawings taken from books and such specimens of human bones as could be obtained in a then remote country village, it is an open question whether the accuracy and beauty of the model or the difficulties under which it was formed are the most to be admired.

That the model was beautiful and accurate is not open to dispute, for it was exhibited at the Society of Arts in the year 1826, and won the first gold medal of the Society; the distribution of the rewards of the Society of Arts having been made at the King's Theatre Opera House on Monday, the 29th of May, 1826. The ticket of admission is now before me, and was signed "Arthur Aikin, Sec." The entrance was the King's private one in the Haymarket.

The history of this model, the difficulties which attended its formation, the hopes and fears which must have been excited in its progress, and the influence it had upon young Towne's career, would, if they could be told, afford as much interest as, and far more instruction than, a novel; but the whole truth upon these topics cannot be obtained, for Mr. Towne was always reticent on these points.

His daughter, however, writes that "The skeleton was commenced without the knowledge of my father's family, worked at in secret and by night with the dim light of a candle. The difficulties he had to contend with may be more easily imagined than described. My father had been engaged upon his work for some months, and the model was near completion, when one night he accidentally upset a heavy piece of furniture, which in its fall broke some glass and caused a terrible crash. The noise wakened his father, who, on a tour of investigation to ascertain the cause of disturbance, surprised his son, and naturally pressed inquiry into the nature and object of his nocturnal work. The following day he re-examined the skeleton and manifested anxiety to assist in any way that might further the views of his son. This offer came too late, the work was almost finished; but after some hesitation my father suggested that he would like to go to the Fitzwilliam Museum at Cambridge, and compare his nearly finished skeleton with one exhibited there. An introduction was obtained to Professor Clarke, who evinced great interest in the boy and his work, and gave the opportunity desired. The kindness of the Professor, and the comparison thus made with an accepted work of art, yielded all the encouragement needed to bring his model to London to compete for the gold medal offered by the Society of Arts. Before taking this step he was anxious to secure a testimonial of accuracy and merit from some high authority, and with this object in view he persuaded his father to let him visit London."

He came to London by coach in 1826, and went to one of the old inns in Bishopsgate Street, where he, a stranger and a boy with a country cut about him, with difficulty obtained a private room, the door of which he could lock, in which to place his valued model. While wandering about London, as a stranger, not knowing whom to call upon or what to do, he knocked in

his dilemma at the door, with a red lamp over it, of some practitioner in Hackney, and sought an interview.

In this gentleman he found a friend who received him kindly, lent him bones by means of which he tested and proved the accuracy of his model, and suggested that he should call on Mr., afterwards Sir Astley Cooper.

He subsequently acted on this advice and, without introduction, called on Mr. Cooper; told him, in his own way, of his work, and asked if Mr. Cooper would kindly examine, at his leisure, the model he had brought for his inspection.

It is needless to dwell upon Mr. Cooper's kindly reception of the boy—though Towne's tongue was ever eloquent on the theme—the interest with which he listened to his tale, and the help he promised him—for it is a matter of history that with Astley Cooper all this was natural, and it is a matter of fact that when young Towne left Mr. Cooper's room, after his first interview, with an invitation in his pocket to breakfast the next morning with the great man and surgeon, and to bring his model, he did so with a light heart and a bounding footstep, and with the conviction that the object of his mission to London—so far as he saw it—was nearly accomplished. But how little he did see!

What passed at breakfast, and at the subsequent examination of the skeleton, Mr. Towne could never relate. He often tried to do so, but never succeeded, for the recollection of the interview excited emotion, and every attempt to relate its events made his voice husky. But this touch of nature was all sufficient, for what better evidence was needed to indicate, that no words could express the encouragement and sympathetic kindness the great surgeon showed to the boy artist, who left the house with a testimonial as to the accuracy of the model he brought for criticism, and a letter of introduction to the late Mr. Benjamin Harrison, the then highly-respected treasurer of Guy's Hospital, by means of which he became connected with the hospital. The testimonial, which is now framed below the skeleton, is as follows:

“I have examined the model of a skeleton made by Mr. Joseph Towne, of which I most highly approve.—ASTLEY COOPER.—February 20th, 1826.”

It should be added, however, that whilst Mr. Cooper was

examining the skeleton, and evidently turning over in his mind the practical outcome of such a work, he observed, "What do you mean to do—to make skeletons till you are a skeleton yourself?" Towne often told this tale and laughed over it, feeling, but not expressing, what others may express,—the satisfaction, that Sir Astley Cooper lived long enough to witness the great success of his young *protégé*.

The letter from Mr. Cooper to Mr. Harrison settled the young modeller's career. For in Mr. Harrison he found a treasurer who had the interest of the medical school over which he presided well at heart, and who appreciated at once the probable value of such a worker as Joseph Towne seemed likely to prove. An arrangement was then and there made between Mr. Harrison and Towne, and on February, 1826, it was completed.

What the arrangement was has never been made clear, for the Guy's records contain no note upon the point, and it is probable that it was more of a mutual understanding between the treasurer and Towne, than any definite contract. All Towne bargained for, was a room to himself upon which he could turn a key when he liked, and the promise of every kind of help from Mr. Harrison, should help be required, in the unknown work he had undertaken to originate.

From that month in 1826 till within two years of his death, that is, for upwards of fifty years, did Mr. Towne work at Guy's. He worked in his own way, was aided by all the resources of the Hospital, which, up to 1853, paid part of his salary, and of the school, which since that date paid all. From John Hilton, then demonstrator of anatomy, he received, for many years, aid in dissections which cannot be over estimated, and from every surgeon and physician he had help and encouragement.

How carefully and skilfully he worked, those who worked with him can alone know, but all who ever walked round the Guy's museum can testify by the magnitude of the work he has left behind to his industry—magnitude, not only in respect to the number of works executed, upwards of one thousand—but magnitude with respect to their importance and scientific value; for there can be no question that as models, whether anatomical, pathological, or cutaneous, they are not only life-

like representations of what they are intended to show, but that as works of art they are as remarkable as they are perfect.

These models, moreover, it is to be recollected, were the outcome of an entirely self-taught genius, the product of a man who, with a high order of intellect, great independence of character, and untiring energy, would have been foremost in any walk of life he had been permitted or had selected to walk.

It should also be stated that Mr. Towne received for a second time the gold medal from the Society of Arts for some models of the brain, and that this attempt to model anatomical subjects was amongst the very first made in this country. The medal was presented to him in April, 1827.

"Dear Sir,—Your model of the brain came under the consideration of the Committee at the Society of Arts this morning. There was, as you may suppose, but one opinion of its merit and value, and the gold medal was voted unanimously.

"This reward must be confirmed by the Society itself on two successive Wednesday evenings, but you need not be in any doubt as to the ultimate result.

I am, dear Towne,

"Yours very truly,

"WM. YARRELL."

"RYDER STREET, ST. JAMES'S;

"28th March, 1827. Two o'clock."

"I have examined," wrote Sir Astley Cooper on February 24th, 1827, "with great attention the models of the brain which have been executed by Mr. Towne, and I feel that I have not words to express my high opinion of their merit."

"The models," wrote the late Mr. R. Grainger on March 3rd, 1827, "are accurate copies of different parts of the human brain they are intended to represent. They are among the very first attempts which have been made in this country to represent the different parts of the human body by wax models;" and it may be added that the series, as completed, forms a unique one of brain anatomy; since it not only includes thirty sections of the organ to exhibit all its parts, but thirty nine models to illustrate its development from the earliest fœtal condition and the arrangement of the cerebral convolutions in the principal varieties of the human race.

It has been stated that Mr. Towne worked for Guy's Hos-

pital for fifty years, and so far as London is concerned he worked for Guy's Hospital alone. He, however, executed many models for abroad, and many fine specimens of his work are to be seen at Calcutta, Madras, Bombay, New York, Alabama, New South Wales, and Russia.

One of his finest works, which he made originally for India, but which after the mutiny was left upon his hands, has been secured for the Guy's museum. It represents the fifth nerve with all its branches, distribution and connections, and is made of some material which will indurate rather than decay by time. It may be regarded as his masterpiece in the way of modelling, and is valued accordingly. He had made likewise, of the same material, a wonderful model of the lower extremity a copy of the one in the Guy's museum, and on this during his last illness he spent many happy days. He looked upon it as his last work for his old institution, and the way he worked at it, and the enthusiasm with which he regarded it, was such as is not often permitted for any one to witness in the aged; but, as he said to the writer of these pages, "I should like to show what stuff the old boy has in him yet; his last work shall not be his worst." Sad to relate, however, this work when just completed was destroyed by the ignition of some spirit which flowed out of an upset spirit bottle that was used at his work. This accident, however, never damped the modeller's ardour or daunted his courage, for as soon as the effects of the mischief had been swept away, the eager and steady hand, the enthusiastic but overwrought brain, returned to their labours with the view to its restoration. It was not, however, so to be, for his strength soon failed, the brain's vigour went, and the model as he left it now remains a noble wreck, with enough beauty in it to speak of its departed glories, and more than enough to show how by the revivifying power of the master modeller it might have been made to tell its wondrous tale.

In 1839, when out of health, and on a recruiting visit to the country, Mr. Towne, with the natural restlessness of an active mind, turned his attention to the subject of the incubation of the egg, and on the suggestion of the treasurer of the Hospital, Mr. Harrison, he prepared a series of models illustrative of the changes which occur in the egg during incubation. He

spent eight months on this work, and certainly not in vain, for the models he made are exquisitely beautiful, and the observations he published on the subject in the 'Guy's Hospital Reports' for 1839, with coloured drawings, contain, what were then considered, many new points. Indeed, I have heard it said, that to Mr. Towne's work many recent so-called authorities have been much indebted. At any rate, I should like to refer the scientific reader of these pages to the paper in question, for it is full of interest and instruction.

Mr. Towne, during the years he worked at Guy's, executed 200 unequalled anatomical models, which were mostly made from the dissections of the late John Hilton; 240 pathological, and 560 of diseases of the skin, the majority of which were selected from hospital cases by that great dermatologist and physician, the late Dr. Thomas Addison. This series includes every known variety, and many very rare forms of skin disease. The models, moreover, are so true and lifelike that the student may imagine, and that without the slightest effort, that he is looking at the original.

The last models Mr. Towne really made were a series, selected by the author of this memoir, to represent the different degrees of harelip, from the congenital cicatrix of the upper lip, to the more complicated double deformity. They were not thoroughly completed at his death, but they are now mounted as he left them and are very good. It is interesting likewise to record, that this series was one, which, as Towne informed the writer, the late Mr. C. Aston Key was very anxious to have had made. By nature Mr. Towne was doubtless destined to be a modeller, for the early evidence of his skill in this art, and the success of his endeavours, even at the age of seventeen, clearly marked this out as the career he ought to follow; but it should be known that as a sculptor he was at least as good, and would doubtless have been equally famous, had time and opportunity lent their aid. The busts of Sir Astley Cooper and Dr. Thomas Addison, now in the Guy's museum, nearly speak to the truth of this opinion, and many other works of a high order still exist to support this judgment. In 1837 he made an equestrian statue of the late Duke of Kent, our present Majesty's father, and on the 21st of October, 1837, he was commanded to attend at Buckingham Palace, and had the honour of an interview

with the then young Queen, when her Majesty was graciously pleased to express her approval of the work, and the statue was deposited in the Queen's private apartment. A little later he made a statuette of the late Duke of Wellington, and there was in the possession of the family an autograph letter from the great Duke testifying to its goodness. Many other busts and monuments were the works of his hands, but no records of them have been preserved. An excellent monument of Bishop Otter was, however, erected in 1844 in Chichester Cathedral.

With respect to the bust of Sir Astley Cooper, it is only right to add, that it was raised to the memory of this distinguished surgeon by his pupils, and opened to public inspection by the late Dr. Richard Bright, on May 10th, 1842, after giving away the prizes to the pupils at Guy's. The opinion concerning it of the late Royal Academician, Mr. G. H. Bailey, is shown by the following letter :

"ASTLEY COOPER'S BUST.

"10, PERCY STREET, BEDFORD SQUARE ;

"*Jan. 14th, 1842.*

"DEAR SIR,—I was much gratified with the sight of your splendid bust of Sir Astley Cooper, and have no hesitation in pronouncing it the finest likeness extant. I should esteem it a great favour if you would allow me to correct the head of my statue, intended for St. Paul's Cathedral, from a cast of it.

"I remain,

"Your obedient servant,

"JOSEPH TOWNE, Esq."

"G. H. BAILEY."

I may add that Mr. Towne found it necessary to give up the work of the sculptor as it interfered too much with his modelling. He had learnt, that for a man to do the one thing of his life well, that is, the business of his life, he must often sacrifice other work. In his case he had to give up what he dearly loved, but it was his duty to do it ; so the task, though hard, was not difficult.

As a modeller the career of the subject of this memoir was remarkable, remembering that he was self taught, and that he had never in his boyhood even seen the art carried out. How he made the first skeleton without good bones to model from remains a mystery, but he did, and it can only be explained by the wonderful power he possessed of reproducing

in material shape from his mental vision whatever subjects he had carefully observed. Thus, the writer of this memoir knows that he from memory had more than once fixed in marble the almost speaking likeness of someone who had gone over to the majority; and startled another, who had never sat for his likeness, by the unexpected vision of his own countenance immortalised in marble in no uncertain language.

On a certain occasion, also, when discussing with Mr. Towne the talent which a great artist had displayed in pouring in bronze the form and figure of what is called the king of animals, I was astonished by his criticism, in which he said, in his own quick way, "they," the animals, "are good in form and in position, but there is no life in them." I went away thoughtful and not quite satisfied, but when years later I by chance took up an animal of Mr. Towne's moulding, and saw there how instinct with life the dead clay was, and how a child of my own on seeing it at once called out, "Why, the giraffe is getting up," I realised at once the truth of what he said, and was enabled to see in the model of the giraffe which I possess the power in man of imparting even to clay, the idea, if not the reality, of living and moving force. I may add that this model was taken from the first giraffe that came to this country, which was exhibited at the old Surrey Zoological Society's Gardens, in 1832. The gardens became, later on, the site of St. Thomas's Hospital, and are now built over.

It was such power as this, which was folded up in him at his birth, and which he subsequently developed. By his thought and industry, he so highly cultivated this power as to enable him to embody in a material shape, forms, figures, colours, and ideas, which less gifted men would never have realised. Can it, therefore, be a subject of surprise that he was a great modeller?

Towne was, however, much more than I have stated, for, as a man of original and independent thought, he was remarkable, and the character of his work probably helped to make him what he was. For it is to be remembered that his work was solitary, and that from the age of seventeen he was compelled to be alone, in his studio, for many hours of each day. Much of his work was also to him mechanical, and as his mind, if I may so express it, was not absorbed in his art labour, it was left free to think and to work out such problems as accident or

whim might suggest. His thoughts consequently were often very original and always suggestive. If they were ever narrow, such a narrowness was to be explained by the nature of the process under which they were nurtured, and by the want of that freedom of intercourse and interchange of ideas with the outside world, which is so necessary to give breadth and liberality to human thought. What Towne's thoughts may have lost in breadth they gained, however, in depth, for they were always well formed, clear, and suggestive.

The late Mr. Hilton, who for so many years spent hours daily with Mr. Towne, when making the dissections from which Towne's beautiful models were made, often in later years spoke of the benefit he himself had derived from his association with the modeller, of the pleasure of that association, and the singular clearness with which Mr. Towne thought and could always express his thoughts. I may add, likewise, that Mr. Towne frequently spoke to the author of this paper of the pleasure and benefit he had himself always experienced from his enforced association with Mr. Hilton. Indeed, a strong feeling of regard and mutual esteem existed between these two very different men, and if from circumstances they were separated as years went on, when the struggling demonstrator of anatomy had developed into the successful surgical practitioner, it is pleasant to know that in the time of the sear and yellow leaf a feeling of that common nature which all living, and particularly human things possess, found an excuse for a renewal of former friendship.

To the subject of this memoir much pleasure was given by the knowledge that time, success, and circumstances had not effaced from John Hilton's memory the pleasures and advantages of a quarter of a century's close friendship, and that in the aged hearts of both there was a spot which was still sensitive to the magic touch of the memory of former years.

In 1858 Mr. Towne delivered at Guy's Hospital a short course of lectures on the Brain and the Organs of the Senses and of the Intellect. He pointed out that the organs of the senses do not themselves perceive, but are merely the intermediate organs for conveying impressions to the brain; and that the nerves, which serve as the conductors between the organs of the senses and the brain, are only capable of im-

parting the sensations for which they are specially provided. The eye is a perfect camera obscura and nothing more. This last idea he developed subsequently in a more expanded way in a series of very valuable and suggestive papers "On the Stereoscopic Theory of Vision, with Observations on the Experiments of Professor Wheatstone," commenced in the 'Guy's Hospital Reports' for 1862, and ending on "Binocular Vision," in the volume for 1870.

These papers display a vast amount of ingenuity, originality, and talent. They are full of striking and novel experiments, and are doubtless valuable contributions both to optical and physiological science. That this is the case can hardly be doubted, when we read in a recent work on 'Sight; an Exposition of the Principles of Monocular and Binocular Vision,' written by Joseph Le Conte, LL.D., and published by Kegan, Paul & Co., 1881, views so strikingly similar to those of our lamented friend.

Mr. Towne was married on September 20th, 1832, to Mary Butterfield, who died in March, 1881, and left several children. He left also some models for disposal, the best of which were purchased for the museum of Guy's, whilst some have since found a home in the University of his county, Cambridge. The family have kindly presented to the hospital the whole of the moulds from which the models have been made,—Mr. Towne having always expressed a wish that such moulds should never be allowed to leave the walls of the hospital for which he had so long, in love, laboured. But above all, Towne left a high reputation for all that is honorable, true, and just. Gifted with special and endowed with high faculties, he improved and utilised each and all. He made for himself opportunities for progress and took full advantage of others as they occurred. He performed the daily duties of his life with singular regularity and closeness, and as a principle never allowed himself to do less than his best. He did all this likewise on a high and yet deeply religious principle. No wonder, then, that success attended his labours, and honour crowned his brow, since with such materials to use, and such motive powers to work with, fortune has never, since the world began, failed to follow endeavour, nor human industry to miss the victor's crown. May we who read this lesson take it to heart, and may the lesson itself help those who read it.

A CASE OF PHOSPHORUS-POISONING

WHICH ENDED IN

RECOVERY UNDER THE ADMINISTRATION OF
OIL OF TURPENTINE.

BY C. HILTON FAGGE, M.D.

As all the other cases of acute phosphorus poisoning admitted into the wards of Guy's within the last few years have proved fatal, the following case, which ended favorably, appears worthy of being recorded. It must not, however, be regarded as proof of the efficacy of oil of turpentine as an antidote, for medical literature contains several instances of recovery after jaundice and other severe symptoms had set in, without such treatment having been adopted.

For example, in vol. xv of the 'Annalen des Charité Krankenhauses zu Berlin,' published in 1869, Schultzen and Riess report four recoveries among ten cases; and besides these they allude to three other successful cases observed during the same period of time, in each of which the early application of the stomach-pump removed almost all the phosphorus from the patient's stomach, but in which, nevertheless, slight jaundice, increase of liver dulness, and tenderness in the right hypochondrium made their appearance. Still, there is sufficient evidence of the value of the remedy to encourage us in continuing to employ it, at least until some alternative method of treatment shall have been suggested. I refer especially to the experi

ments of *Personne*, of which an abstract appeared in the 'Comptes rendus' for 1869. Of fifteen dogs, five to which phosphorus alone was administered died; whereas, all but two of the other ten recovered, they having had turpentine given to them in addition, either at once, or an hour or two later than the poison. Another series of experiments has since been made by J. Rommelaere in Belgium in 1874. The first suggestion of employing oil of turpentine in acute poisoning by phosphorus is due to Andant of Dax. In 1868 he met with the case of a man who, having taken phosphorus with suicidal intent, afterwards swallowed oil of turpentine by way of making doubly sure of killing himself, with the result that he suffered but little or not at all. A detailed paper by Andant may be found in the 'Annales d'Hygiène' for 1873. In the meantime the question was taken up in Germany by Köhler, who published a work to which I have not been able to refer. His great point appears to be that the only kind of oil of turpentine likely to be useful is that which has not been rectified. Recent medical literature seems to contain little further information, but Naunyn, writing in Ziemssen's 'Handbuch' in 1876, expresses himself with reserve. The dose of turpentine which he recommends is two or more grammes every quarter of an hour until at least ten grammes have been taken.

For the following excellent report of the case I am indebted to my clinical clerk, Mr. E. Stanley Tresidder.

CASE.—George P—, waterside labourer, admitted into Philip Ward, under Dr. Hilton Fagge, February 23rd, 1882, in consequence of having swallowed half of a sixpenny bottleful of "Cooper's phosphorus paste" for destroying vermin.

Family history.—Father, æt. 70, living; mother died of old age, but was subject to fits. One brother healthy.

Personal medical history.—Has never before been ill. Never had syphilis. Two years ago suffered from hæmorrhoids. Is married and has five children. Until two years ago was accustomed to drink moderately of beer only. Since that period has taken to whiskey, and has generally been drunk about twice a week.

History of present illness.—At 3 p.m. on 23rd February, not having previously contemplated suicide, he mixed half a sixpenny bottle of "Cooper's phospho-paste," sold for destroying

vermin, with some whiskey and drank it. He says he did it to frighten his wife. His pecuniary circumstances are good. He applied to a local medical man and vomited in his surgery, and soon after (at 5.30 p.m.) came to Guy's Hospital, where he was at once admitted for treatment, and had immediately an emetic of—

R. Zinci Sulph., ʒss,
 Vin. Ipecac., ʒvj,
 Vin. Antimonialis, ʒij,
 Aquæ ad ʒij.

To be taken in six ounces of warm water—

and vomited. The vomit smelt strongly of phosphorus and had a white opaque vapour floating over it. He has been drinking hard during the last five days, *i.e.* has been continuously drunk since February 18th.

Condition on admission.—Low and depressed, pale, skin cold. Pulse good. Influence of alcohol still obtains. Breath and clothes have a slight alliaceous odour but not strong. Acute and severe pain in epigastrium. Burning sensation along the course of the œsophagus, which gives him the sensation of impending death.

Treatment and progress.—On admission into Philip Ward as No. 28, another emetic was given. The vomit still smelt of phosphorus. The vomiting continued until about 2 a.m., and then had no smell of phosphorus. Hot milk was at first given at intervals, but about 2 a.m. he took some cold milk. Pain in epigastrium much better.

R. Magnesiæ Carb., ʒss,
 Mucilag. Acaciæ, ʒiiss,
 Olei Amygdalæ, mx,
 Syrupi, ʒss,
 Aq. ad ʒj. Secundis horis.

February 24th, a.m.—Pulse 90; says he is comfortable, except that occasionally pain in epigastrium recurs; it is relieved by food but increased by pressure. Liver not enlarged nor tender to touch. No pallor of face.

Noon.—Pulse 80, temp. 99·4°. Dr. Stevenson saw patient this afternoon with Dr. Fagge. Symptoms few, except the pain at epigastrium; does not complain. Former medicines discontinued and the following prescribed:

℞ *Magnesiae Carbonatis*, ʒss,
Ol. Terebinthinæ, ℥xv (American),
Mucilaginis Acaciæ, ʒij,
 Aquæ ad ʒj. Om. horâ.

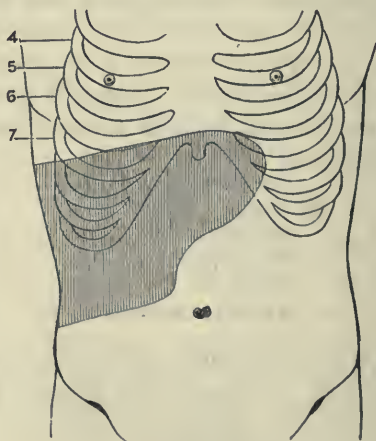
Even.—Pulse 80, temp. 99·4°. Bowels not moved during the day.

10 p.m.—℞ *Mucilag. Acaciæ*, ʒvj,
Liq. Bismuthi, ʒiss,
Syrupi, ʒiss,
 Aq. ad ʒj.

M. One ounce to be taken every hour. Rep. mist. *Mag Carb. cum Terebinth.*, 4tis horis.

25th.—Comfortable; does not complain except of fulness; says he is filled up by the medicine. Bowels moved once last night, the motions dark, not abnormal. Dulness to-day over liver shows increase in its size. Urine scanty.

℞ *Ol. Terebinthinæ*, ℥xxx,
Ol. Menth. Pip., ℥j,
Magnesiae Carb., gr. x,
Mucilag. Acaciæ, ʒij,
 Aq. ad ʒj. Om. horâ.

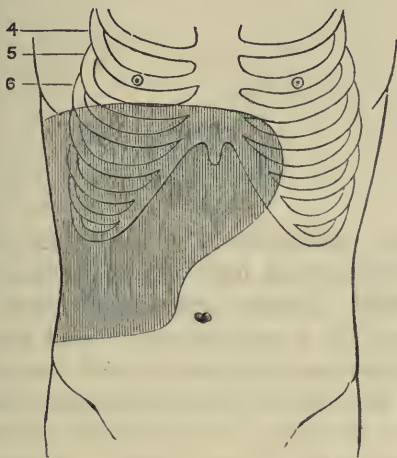


Feb. 25, 1882.

26th.—During night has been purged by the medicine. Internal hæmorrhoids have come down and have now been returned within sphincter. Pain of hæmorrhoids relieved by

Supp. Morphiae. Injection of morphia gr. $\frac{1}{5}$ subcutaneously, given at 2 a.m. Rep. Mist. 3tiis horis. Pupils are now contracted from the effects of the anodyne. Skin slightly yellowish-brown, but his wife pronounces it not abnormal. No pain. Dulness of liver extended slightly since yesterday. Tongue dry and brown at dorsum, red at tips and edges. Complains of thirst and feels hungry. M. temp. 100° . E. temp. 101.4° .

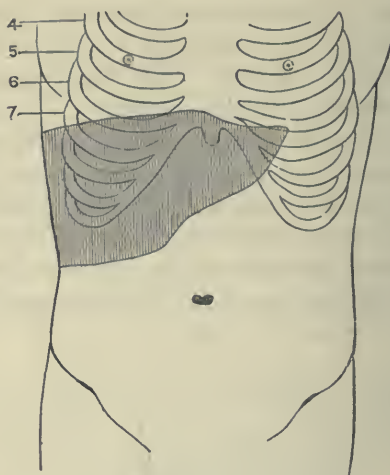
27th.—Bowels moved twice last night. Has intense headache, which first occurred during the night, and was a little relieved by an ice-bag. Dulness of liver still more increased, and now there



Feb. 27, 1882.

is tenderness on pressure. Skin slightly yellow and conjunctiva tinged. Tongue moist, slightly furred. Bowels moved once during the day. Urine dark brown, no reaction for bile pigments with HNO_3 or Iodine test. Pulse 88. M. temp. 99.8° . E. temp. 100° .

28th.—Patient this morning looks thinner, paler, and more yellow; yellowness well marked in conjunctivæ. Bowels moved once, the motions being semi-solid, light clay-coloured; blood coloured fluid in pan from piles. Area of liver dulness decreased; it is not so high up as it was yesterday by one inch, nor so low down by an inch and a quarter. During night was unable to pass urine. No. 9 catheter passed and a pint and a half of



Feb. 28, 1882.

dark-brown urine withdrawn. Sp. gr. 1025. Acid reaction. Treated with HNO_3 on a white plate, a very faint play of colours is observed. Tongue coated on dorsum. Pulse 60, feeble. At 3 p.m. Dr. Fagge saw patient and discontinued turpentine, in consequence of its being stated that the urine contained a small quantity of blood, which fact, however, subsequently appeared to be open to considerable doubt.

March 1st.—Pulse 64. M. temp. 98.8° . Patient seems better, not very drowsy, is able to read and feels better. Bowels moved last evening, solid, of a drab colour, blood from piles surrounding it. Area of liver dulness less to-day. Has had during last two days a dry, troublesome cough. Tongue moist and clean. No ecchymoses are visible on body.

2nd.—Is much jaundiced to-day, of a greenish tinge, otherwise much as yesterday. Face looks dull, and there is some drowsiness, but intellect is clear. Slept last night during short intervals, and has been dozing occasionally during the day. No headache. Urine still dark, sp. gr. 1032. Bowels moved, the motions light in colour. Area of dulness over liver decreased, but tenderness great, the slightest pressure or percussion giving pain. Tongue clean. Pulse 52, small. M. temp. 99° .

3rd.—Much as reported yesterday. Liver dulness about the

same. No perceptible change in colour of skin or conjunctiva. Pulse 56, not strong. M. temp. 98.6° . E. temp. 98° .

4th.—Improved. Jaundice less, conjunctiva clear. Pulse 52. M. temp. 98.2° . E. temp. 97.8° .

5th.—Patient left the hospital to-day in opposition to advice of house-physician.

6th.—Patient returned to hospital to-day to see Dr. Fagge. Was better. Jaundice less.

7th.—To-day I went to the patient's house and saw him. He was up and had been out a little, seemed cheerful; said he felt weak and unable to work yet; was tired after standing and got pain in the liver, which went off after resting in recumbent position. Liver still tender on pressure. Jaundice less. Pulse 64, full and strong. Takes his ordinary food, but has taken no spirits yet.

9th.—Patient came to-day to Philip Ward. Seemed pretty well; seemed to fear being detained, so left before an examination of his case was made.

23rd.—The patient was again visited at his home; found apparently in good health. He has resumed work, feels rather weak, unable to do as much as heretofore. Still abstains from spirits. Tenderness over liver gone, no enlargement; dulness now measures four inches in the mammary line. Skin still of a dark colour, which patient says is normal.

A CASE
OF
SYMMETRICAL SOFTENING OF THE
CORPORA STRIATA,
FOLLOWED BY
BILATERAL DESCENDING DEGENERATION WITH
SECONDARY ANTERIOR POLIOMYELITIS.

BY W. HALE WHITE, M.D.

OWING to the kindness of my colleague, Dr. Frederick Taylor, I am enabled to publish the following case, which came under my notice as Resident Medical Officer of the Evelina Hospital:

W. C—, æt 6½, a boy, was admitted under Dr. Taylor on March 24th, 1881. The family history was very good. The present illness dated from the January previous, when, during the exceptionally cold weather we had then, the boy played the truant, leaving school in the middle of the morning without permission; he was next found lying in the snow, and when picked up could not stand, the right arm and leg being partially useless. On arriving home some improvement was noticed, for he could then walk, although he dragged the right leg. Before this attack he was a remarkably intelligent boy, but after he was found in the snow he never spoke or read anything. Incontinence of urine and loss of power

over the rectum came on at the same time, and have continued ever since, although latterly not so much as at first. The father told me he was not surprised at this attack, as he had had the child's head examined by a phrenologist, who thought there was something wrong, and said the child must not be allowed to study too much.

On admission.—Is a fat, well-nourished child; heart and lungs healthy. He never speaks, but seems to understand a little when spoken to, and also to take slight notice of surrounding objects; nurse says he is certainly "stupid," he always cries when any one goes near him, and always lies upon his right side. When he is in bed there is no obvious paralysis of any part of the body, but on attempting to walk he stumbles, holding the right leg stiffly, and scarcely putting either sole on the ground; he carries the right arm flexed at the elbow, with the hand drooping, and prefers to put the left forward for use although he can move the right. Patellar and plantar reflexes are well marked, cremasteric reflex and ankle clonus absent. He passes everything under him. There is no vomiting, no tache cérébrale, nor optic neuritis, although the discs are very red.

April 24th.—Up to this date he has remained in the same state as on admission, lying upon the right side, and passing urine and fæces under him. The temperature has been normal or subnormal. This morning he suddenly began to cry violently, and nurse says he had a fit, but when I saw him a few minutes after he was lying still, screaming violently, all the limbs were quite rigid, and there was a total absence of all reflexes. This crying continued for some hours, and the temperature rose to 101.2° .

26th.—The child is lying on his back with a vacant expression, and constantly moving his eyeballs and looking up to the ceiling. He does not seem unconscious, but will not speak. There is rigidity of all the limbs except the right leg, and he can move them all except the right arm. Reflexes are all absent; there is slight tache on the legs; the cheeks are flushed.

May 1st.—There is no change in the condition last noted; the nostrils have to be syringed with Condyl's fluid in consequence of a sero-sanguineous discharge.

5th.—Refuses all food but milk; very fretful, otherwise the same.

15th.—Lies in much the same condition; the head is generally but not always severely retracted.

26th.—During the past three weeks there has been much divergence of opinion as to the existence of optic neuritis; as this was probably due to the extreme difficulty of the examination owing to the restlessness of the eye, chloroform was given, and it was finally decided there was no optic neuritis, but only extreme vascularity.

27th.—Nothing unusual was noticed in the child's condition last night, but on taking the evening temperature at 9 p.m. it was found to be rising; this it continued to do till 2 a.m. this morning, when it stood at 106.6° . It remained between 105° and 106° all night, and keeps up this morning in spite of sponging with vinegar and water. He is completely insensible, the rigidity of the limbs has increased, and he refuses all food.

28th.—Is in exactly the same condition as yesterday, except that the temperature is lower, having only once reached 103° during the last twenty-four hours.

29th.—In the same condition, unconscious and limbs rigid. Teeth firmly clenched, refuses to swallow, is therefore fed with enemata. Temperature often rises over 103° , and when it does so the child is sponged.

30th.—Last night the child suddenly screamed loudly; this was followed by convulsions of all the limbs and face. The abdomen and thighs became covered with red patches. Temperature still high.

June 1st.—Has had several convulsions.

4th.—He is frequently convulsed, the face being chiefly affected; when these attacks commence he becomes exceedingly flushed, and the temperature rises considerably (*i.e.* 104°), and he is pungently hot. The arms are contracted, so that the forearm, hands, and fingers are all flexed. The jaw is tightly contracted; the legs are only slightly rigid.

6th.—Temperature fell this morning to 98° . There is no rigidity of the legs, but it is more marked in the arms.

8th.—The enemata being no longer retained, he is fed by means of a catheter passed down the œsophagus. The jaw is so tightly clenched that it has to be opened with a gag. Temperature still 98° . General condition the same.

10th.—Temperature again rising, having once to-day reached 103.2° . Is getting slowly weaker.

12th.—The child is now frequently sponged, as the temperature often goes over 103° . For some time past the pupils have been widely dilated, although it is some time since any atropine has been used; they respond to light. There have been no convulsions lately.

15th.—Lies unconscious, occasionally crying out; is much weaker. Temperature often over 103° . Still fed with the catheter.

19th.—He died early this morning. He lay quite quiet some hours before death, but towards the end the breathing became very laboured. The temperature rose to 107° .

The eyes were examined by Dr. Brailey just before death, and it was his opinion that there was slight commencing optic neuritis. During his stay in the hospital he wasted very much, and the emaciation was so general that it was difficult to say that any muscles in particular were affected. Throughout the illness he was treated with large doses of iodide of potassium.

Post-mortem examination (eight hours after death).—Rigor mortis well marked. The brain was remarkably firm, vessels and vascular spaces were larger than normal. There was no evidence of tumour, meningitis, tubercles, or disease of the cerebral arteries. Near the anterior extremity of each corpus striatum, just above the anterior commissure, was a brownish patch, about a quarter of an inch in diameter, so soft that it left a hole when cut; these patches were symmetrical, both in colour, size, and position. Well marked grey discoloration was present in both crura cerebri at the anterior inner margin; this tract of degeneration was also very distinct in both pyramidal tracts, especially the left; in the cord there was marked degeneration in the right postero-lateral column, which could be traced up to a communication with the degenerated tract in the left brain. This degeneration of the cord extended down to the lumbar region. There was also some degeneration in the cord on the inner margin of the left anterior column. All the other organs of the body were perfectly healthy.

Appended is an analysis of the temperature which, during the last eight weeks of his illness, was taken every hour.

March 24th to April 3rd.—Temp. ranged from 95° to 96° .

April 3rd to April 17th.—Temp. ranged from 97.4° to 98.4° .

April 17th to April 24th.—Temp. ranged from 95° to 97.4° .

April 25th.—Morning temp. $101.2.2^{\circ}$, evening 104° .

April 26th.—Morning temp. 97.4° , evening 99.2° .

April 27th.—Subnormal all day, 97° .

April 28th.—Morning temp. 98° , evening 100° .

April 29th to May 25th.—Temp. ranged from 96° to 99° .

May 26th.—Morning temp. 100.2° , evening 106.6° .

May 27th.—Morning temp. ranged about 105° in spite of sponging, evening kept at about 102° , if over 103° he was sponged.

May 28th.—Kept at about 102° by dint of sponging whenever it rose over 103° .

May 29th.—Morning temp. 102.4° , evening 99.2° .

May 30th.—Morning temp. 105° ; convulsions; spots appeared.

May 30th to June 5th.—Kept at about 102° by sponging if over 103° .

June 6th to June 8th.—Temp. ranged from 98° to 100° .

June 8th (evening) to June 11th.—Morning temp. averaged about 102° , going once over 103° .

June 11th to June 16th.—Temp. averaged higher, it was more often necessary to sponge to keep it under 103° .

June 17th and 18th.—Temperature ranged higher still, often going over 105° in spite of prolonged and frequent sponging, and finally just before death rising to 107° .

It will be seen from this that we may arrange the exacerbations thus:

March 24th to April 24th.—Temp. normal or subnormal.

April 25th.—First exacerbation, highest temp. 102.4° .

April 26th and 27th.—Temp. normal or subnormal.

April 28th.—Second exacerbation, highest temp. 100° .

April 29th to May 25th.—Temp. normal or subnormal.

May 26th to May 28th.—Third exacerbation, highest temp. 106.6° .

May 29th.—Temp. 99.2° for a few hours.

May 30th to June 5th.—Fourth exacerbation, highest temp. 105° .

June 6th to June 8th.—Temp. normal or a little over.

June 8th to June 18th.—Fifth exacerbation, highest temp. 107°.

Microscopical examination of the cord showed the following changes:—In the cervical region was well-marked degeneration of both crossed pyramidal tracts, especially the right. This differed in no way from that usually described, and invaded the contiguous grey matter of the posterior and outer part of the anterior cornua, but not enough to destroy the cells extensively, although it appeared as if some of them were slightly implicated. In the dorsal region these two degenerative tracts became less and less, although they remained well marked at the lower dorsal part of the cord, where the lowest section was taken. In the upper part of the cord the right direct pyramidal tract was affected slightly; this diminished from above downwards, there being but the merest trace of degeneration in the lower dorsal region. The two patches of softening in the corpora striata were so difficult to examine microscopically that the attempt to get complete sections of them ended in failure.

The first point to be settled was whether this was a case of primary lateral sclerosis spreading into the anterior cornua, and thus becoming a case of amyotrophic lateral sclerosis, or whether all the descending degeneration was due to the patches of softening in the corpora striata.

Primary lateral sclerosis is a very rare affection in the wards, and still more rare on the post-mortem table; and although this was not a simple case of disease confined solely to the lateral columns, the following references may be useful. Others will be found in the many writings of Charcot, and in the article by Erb in 'Ziemssen's Encyclopædia.'

Up to the present, only one case has been recorded of a microscopical examination of the cord in this disease.¹ Probably the majority of the cases which during life are diagnosed as lateral sclerosis or "tabes dorsalis spasmodique" are not so; in fact, Richlin,² who in 1878 published a good epitome of all that was then known on the subject, is very sceptical as to whether there is such a disease as idiopathic lateral sclerosis, and certainly then his doubts seemed justified because of two post-

¹ 'Trans. Internat. Med. Congress,' 1881, vol. i, p. 407, Case by Dreschfeld.

² 'Gaz. Méd. de Paris,' 1878, No. 27 *et seq.*

mortem examinations that had lately been made on cases that were supposed during life to be suffering from this disease ; one, published by Pitres,¹ was found to be a case of disseminated sclerosis, and another, by E. von Stoffella,² was so unsatisfactory that no dependance can be placed upon it. Cases recorded without post-mortem examinations are extremely numerous in medical literature, but they ought to be received very guardedly ; amongst others are the following :—Reinhard von der Velden³ gives a case of a lunatic suffering from spastic paraplegia, in whom the disease could be distinctly traced to his getting wet in an attempt to commit suicide by drowning. He, however, got well in about sixteen months under chloral. Henck⁴ also gives a case due to cold that got well. Other authors giving cases that more or less resemble this disease are Schultz,⁵ Leyden and Westphal,⁶ Richter,⁷ Gee,⁸ Abercrombie,⁹ Berger,¹⁰ Nothnagel,¹¹ Betous,¹² Türck,¹³ Charcot,¹⁴ Erb.¹⁵ Of these cases the three recorded by Gee are interesting in reference to the case now under consideration, as they occurred in children ; but with the exception of the fact that they had spastic paraplegia they differed in all points from it. Türck's cases were recorded as long ago as 1856, but he cannot claim the discovery of the disease, as he appears to have paid very little attention to them. A certain proportion of those above referred to were undoubtedly functional, and it seems to me that all examples of lateral sclerosis that get well are so. I have only seen one patient who is allowed

¹ 'Revue Mensuelle de Médecine et de Chirurgie,' 1877, No. 12.

² 'Wien. Med. Wochenschr.,' No. 21 and 22, 1878.

³ 'Berl. Klin. Woch.,' 1879, p. 563.

⁴ Ibid., No. 3, 1879.

⁵ 'Arch. der Heilkunde,' vol. xviii, p. 352, 1877.

⁶ 'Berl. Klin. Woch.,' 1878, 1879. Leyden, 'Arch. f. Psych. und Nervenkr.,' viii, p. 641. Westphal, 'Charité Annalen,' 1878.

⁷ 'Deutsch. Arch. für Klin. Med.,' vol. xviii, p. 365.

⁸ 'St. Bart. Hosp. Reports,' 1877.

⁹ Ibid.

¹⁰ 'Deutsch. Zeitschrift für Prak. Med.,' 1877.

¹¹ 'Arch. für Psych. und Nervenkr.,' vi, p. 336.

¹² 'Étude sur le tabes dorsal spasmodique.' Paris, 1876.

¹³ 'Sitzungsberichte der Kaiserl. Academie der Wissenschaften, zu Wien,' t. xxi, p. 112.

¹⁴ 'Maladies du Système Nerveux,' 1873, and many other publications.

¹⁵ 'Ziemssen's Encyclopædia,' Eng. Trans., vol. xiii.

by all who have seen her, since the discovery of the disease, to have idiopathic lateral sclerosis. The illness has now lasted twenty years, and she presents every symptom mentioned by Erb and Charcot, namely, the spastic gait and paraplegia extending slowly to the arms, slight tremblings, exaggerated reflexes, and ankle-clonus. She enjoys excellent health except that from inability to walk, she is weak; there is no rise of temperature or trouble with the bladder. The only symptom she has which is not mentioned by Charcot or Erb is optic nerve atrophy, and I have been told by one who has seen several of these cases that this is not uncommon. She was seen by an eminent London physician before the discovery of the disease, and he gave it as his opinion that it was a case of locomotor ataxy. Bromide of potassium, strychnia, phosphorus, Calabar bean, and electricity have all been tried without success. The latter had to be discontinued because whenever the electrodes were applied a gangrenous sore appeared, which was extremely difficult to heal.

Amongst these various references I am unable to find any which would lead one to think that this boy had primary lateral sclerosis, extending from the corpora striata, where it caused softening, to the lumbar region, and which spread into the anterior cornua secondarily; in fact, authors seem to look upon the absence of such a complication as one of the essential features of the disease; for example, Richlin¹ sums up thus:—"En un mot le diagnostic de la paralysie spinale spasmodique se fonde surtout sur l'isolement des deux symptômes, parésie et contracture." Charcot and Erb say the same, although the latter in one part of his article suggests that bladder complications may be due to an extension into the anterior cornua, but he gives no case. Richter² makes a similar suggestion. Russell³ has recorded three examples of spastic paraplegia in two of which he concludes that this occurred, for they both presented bladder symptoms, and one had also high temperature, but in none was there a post-mortem examination. The following reasons will, however, I think, be sufficient to prove that this is not such a case.

In cases of idiopathic spastic paraplegia the disease begins in

¹ Op. cit.

² Op. cit.

³ 'Med. Times and Gazette,' 1880, vol. i.

the lumbar region and spreads up; the microscope revealed the reverse here. It is reckoned by years instead of months, and it occurs in adult life so much more frequently than in children that Erb is led to hint that if it be found in early life it is due to some congenital affection of the cord.

The next possibility is that this was a case of amyotrophic lateral sclerosis, but against that view is the fact that the anterior cornua are not much affected, and also that there was no wasting when the boy came in, although there was rigidity; for in that disease the wasting comes before the rigidity. I therefore believe the true explanation of the child's symptoms to be that he had two symmetrical patches of softening in the corpora striata with secondary descending degeneration, which extended into the anterior cornua. Charcot says he has often observed symptoms which led him to infer that this extension sometimes took place. Symmetrical patches of cerebral softening are not, however, frequently met with.

Dr. Frederick Taylor¹ has recorded a case in which there was degeneration of the posterior half of the cerebrum on both sides, the corpora striata were but little affected, there was well-marked secondary degeneration extending on both sides, through the crura cerebri, pons, medulla, and cord. It is interesting to note in the two cases the following points of similarity. The patients were about the same age, namely, six years, both were boys, in both the illness was attributable to a fall, head symptoms were present, and the course was acute, eleven months in Dr. Taylor's, and six in mine, the cerebral lesion was bilaterally symmetrical, and lastly, it was of a degenerative nature, although in Dr. Taylor's this produced "a brown, jelly-like material," and in the one under consideration it resulted in two patches of softening. In the former, difficulty of swallowing is noted, although it is not stated whether it is due to spastic rigidity of the jaw as in the latter. The view just taken will explain the bladder symptoms, and as regards the rise of temperature extension of the inflammation into the grey matter may have implicated a heat-centre, or if not it must be remembered that anterior poliomyelitis is accompanied by elevation of temperature, although not usually of so irregular a type. The rigidity of the jaw was I suppose owing to the affection of its muscles with spastic

¹ 'Guy's Hosp. Rep.,' vol. xxiv, 1879.

paraplegia, and this leads one to wonder whether in bilateral descending degenerations the muscles supplied by all the cranial nerves become affected as those supplied by the motor fibres of the inferior maxillary nerves did in this case. Theoretically one would suppose that they would, and possibly also the muscles of respiration are affected. We know that the abdominal muscles become rigid, and therefore we may conclude that the intercostals do also. It therefore seems to me that one reason why bilateral descending cases terminate more rapidly than the ascending ones is that in the former the vital muscles, *e.g.* the diaphragm by means of the phrenic nerve, become affected more early than in the latter. The majority of cases of descending degeneration from disease of the brain are unilateral, and in these the same theory that is used to explain the absence of the paralysis of the muscles of respiration in hemiplegia will serve to explain also the absence of spastic paraplegia.

As, however, in this case the grey matter was implicated, one need not depend upon the affection of vital muscles with spastic paraplegia to account for death, for it is extremely probable that it was due to the inflammation extending into some important grey centre, and also that the wasting of the muscles observed all over the body was partly due to the same cause, although it is difficult to say much about that, as the child took so little food.

EXOPHTHALMIC GOITRE WITH MENTAL DISORDER.

By GEORGE H. SAVAGE, M.D.

IN the subjoined paper I have only recorded cases in which Graves' disease was associated with mental disturbance of a marked kind. We asylum physicians are perhaps a little too much inclined to make much of mental disturbance when it occurs with other disorders of the body, but, on the other hand, I think general physicians err in the other direction by not taking sufficient notice of the mental aspect of general disease. If delirium occur it is noticed because it asserts itself, but the slighter changes in disposition and character are passed over. Many diseases, if not all, have their special nervous aspect, and this aspect is more clearly defined in the cases seen in an asylum than in those in a general hospital. Every physician is used to the hopefulness of the phthisical patient and the low spirits associated with digestive trouble; in an asylum the same symptoms are seen in similar cases, only they are much more marked, especially in the latter.

With this introduction I would add that with Graves' disease from the first it was noticed that there was marked nervous disturbance. It occurs generally in women, and generally in young women, and we might at once expect that there would be a nervous colouring to any affection occurring under such circumstances.

Besides the cases of true and complete Graves' disease with

mental disorder I have to record some cases in which, with mental disorder, one or more of the symptoms of this disease were present.

The disease itself requires no description from me, and I would only say that in my experience it is not uncommon to see partially developed Graves' disease which may either be recovered from or may pass into the complete state.

I do not think any good purpose will be served if I write a long paper on the bibliography of this disease or on the insane relations which have been described by others. Suffice it to say that though few cases have been described of fully-developed insanity with this disease, yet several have been observed by both English and foreign authors. Soon after its first description by Graves, Dr. Laycock considered it carefully and wrote several interesting articles in the 'Edinburgh Medical Journal,' in which he fully recognised that with exophthalmic goitre there might occur epilepsy, hysteria or insanity. Dr. Morell Mackenzie described a case with insanity in the 'Clinical Society's Transactions' for 1868, and Dr. A. Robertson recorded one in the 'Journal of Mental Science' for 1875. German writers were careful in reporting similar occurrences. But there are still a few only to be referred to, and I look upon the subject as one that is especially interesting from the close association of the bodily and mental disturbances.

We have got so used to talk of the disease as one of the sympathetic that it will not be without use if we are made to reconsider the data upon which we found this opinion.

The sympathetic may have something to do with the disease, but I for one believe we shall find that the higher centres are not free from suspicion.

In one of the cases in which a post-mortem was made I found the supra-renal capsules disorganised; this is of interest, and may be of use to explain further the functions of those bodies.

I shall now proceed to give the cases which have been under my care, and which form the basis of this short article.

In the first place I shall consider these three cases of exophthalmic goitre, and then deal with certain other cases of mental disorder which have occurred in the hospital, and in which, although there has been no true disease of this kind, one or more of the symptoms common in the disease were present, so

that, although we have not the complete picture, we have a sketch so like it as to make one believe that there is some similarity in the conditions producing both the mental and the physical disorder. I shall not spend much time in investigating the causation of this disease. I shall have to refer shortly to the autopsies in the cases of two of my patients, and then to point out that nothing was specially noteworthy in the condition either of the nervous centres in general, or in the sympathetic ganglia, so far as examined, in particular. By exophthalmic goitre I understand the disease described both by Graves and by Basedow and named after them respectively—a disease in which the most marked symptoms are prominence of the eyeballs, enlargement of the thyroid gland, and rapidity of the pulse. I have now seen several cases in which one or other of these symptoms preceded the others by an indefinite time, so that the disease under discussion began in one case with unusual rapidity of pulse, in another was marked by prominence of the eyeballs, and in a third was first noticed in the prominence of the thyroid body. It seems to me that in some cases the disease runs its course without all these symptoms being present at any time—that the exophthalmos or the rapid pulse may be the chief or only marked signs. If I had to fix upon the most characteristic symptom of the disease, I should be inclined to say that in my experience rapidity of pulse was the most common. In the first cases that I have to describe, all the symptoms were present, and thoroughly characteristic. The thing that struck me most, and at first, was the eyeball prominence, but at once, on investigating the pulse, I found that it was rapid, and on further examination the throat was discovered to be unusually large. The three cases to which I am about to refer are alike in occurring in young women, and in two of them there was strong neurotic inheritance. One case had an insane father, another had insanity and eccentricity on the maternal side, and the third had an insane cousin. In two of the cases a fatal result has followed, and I fear that we shall only have to wait a comparatively short time before the youngest and last case follows the others. In the end, these patients suffered from visceral troubles, diarrhoea being the proximate cause of death in two. Before making further remarks upon the cases in general, I shall briefly give the history of each case.

CASE 1.—C. S—, single, 28, admitted in 1877, an artist, having two insane relations. This was the first attack of unsoundness of mind, the cause being unknown. Two months before admission into Bethlem she became incoherent, was noisy and excitable, had delusions, thought that she was an actress, and had false ideas of her power and influence. The chief characteristics of her insanity were excitement, incoherent talking, violence, destructiveness, and sleeplessness. She had suffered a good deal from palpitation of the heart, and according to her friends, exophthalmic goitre had been developing for about a year, and she had worried herself considerably about the change in her personal appearance. On admission she was found to be a pale, anæmic girl, with marked and pretty uniform protrusion of both eyeballs, and slight enlargement of the thyroid gland. The number of the pulse was not then recorded, but later it was 140 to the minute. She continued to be acutely maniacal during the next month, became filthily dirty in her habits, constantly filling her mouth with dirt, stones, and sticks. She was treated medically with liquor ergotæ and tinctura belladonnæ, but without any benefit. Within ten weeks of her admission she became much weaker, and was confined to her bed. She suffered much from vomiting and purging; the pulse was 135, respirations 40, temperature 98°. The eyes were more prominent than ever, especially the left; the pupils, which at first were large, became somewhat smaller; she was still dirty in her habits, but less noisy; palpitation of the heart was marked, and a systolic bruit was heard at the base of the heart. This was very rough in character, but was not audible at the apex. A bruit also occurred at the base of the neck on both sides. The patient now became dull and sleepy, the skin hot and dry to the touch, although the temperature was really normal. She became more dull and sleepy, and could hardly be roused. Pulse 140, respirations 50, temperature 100. No lung complications could be detected; heart palpitating; conjunctiva suffused, optic discs pale, vessels appeared large and dilated; the vomiting had become less, purging continued. On the next day she was reported to be gradually sinking. Before death she recognised her father, but this was the only sign of mental recovery throughout her stay in Bethlem.

Post mortem.—The brain was found to weigh 44½ oz., the

dura mater was thick but free, the arachnoid thin and quite normal. There was an appearance of congestion about the finer vessels, giving a bright red dendroidal appearance to the surface of the brain. There were no local wastings of the convolutions; there was slight excess of subarachnoid fluid, grey matter normal but thin; white matter soft; ventricles with excess of fluid, walls and floors of ventricles granular, vessels at base normal; sympathetic normal in appearance; lungs congested at base, and having several patches on the surface like apoplexies. Heart $13\frac{3}{4}$ oz., firmly contracted; early atheroma visible in the aorta; clot in the right auricle. Both kidneys congested, capsule thick and adherent; liver very fatty; supra-renal capsules small and breaking down. There was some fulness in Peyer's patches, and the spleen was normal. On careful investigation of the cervical ganglia of the sympathetic, perfectly healthy appearances were found; in fact, I have never examined a more typically healthy sympathetic in my life. The case, then, so far as the post-mortem is concerned, exhibits no marked disease except in the supra-renal bodies. The congestion in Peyer's patches was remarked upon from the fact that similar conditions have been described by Mr. Howse, in the 'Pathological Transactions' for 1877, as having been found in another case of this disease.

CASE 2.—A. J B— was admitted in July, 1879. Her father had been insane; a sister died of phthisis. She was single, twenty-four years of age, and engaged in a paper manufactory. This was the first attack of mental unsoundness, and had lasted one month before admission, the predisposing cause being said to be inheritance, and the exciting cause grief at the death of her sister. The earliest symptom noticed was melancholia. This gradually became more marked, and the patient at last became dull and unoccupied. This depression passed off, and she became very excited, and before admission she was described as being in a state of great excitement, howling, singing, and dancing alternately. She was under the delusion that she had committed some great crime, and talked to imaginary people. Her mother said that she was constantly talking, and refused her food. She is described then as having staring eyes, and it was said that she was in great dread, and started at the smallest

noise, almost declined to speak, or only said a single word at a time. She wrung her hands and trembled when any one approached her. Since the death of her sister she had been much excited about religious matters. Her general health had been fair up to the time of admission. The catamenia were regular. After the announcement of her sister's death she became profoundly depressed, and then acutely maniacal.

On admission she was described as having a pale, pimply face; exophthalmic goitre, with a pulse of 140. The first cardiac sound is described as a slap, the second being almost inaudible. There was a well-marked *bruit de diable*; and bronchial breathing, with mucous râles at the left apex. She had some cough. She did not answer questions, and was in the habit of slipping from her chair on to the floor, and lying there. She was unoccupied, dirty in her habits, refused her food, would not dress herself, and was generally obstinate. She had to be fed, within a week of admission, with the stomach-pump, became furiously excited, and bruised herself so much that she had to be confined in the padded room. The stomach-pump was continued for some days, but in the end she took her food fairly well. Twelve days after admission diarrhoea and sickness came on; the pulse became very feeble, though she at the same time became very excited. She slowly lost strength, and, though fed by nutrient enemata, she sank and died sixteen days after admission.

Post-mortem examination showed a brain of 46 oz., very soft, but the membranes generally and intensely congested; the lungs exhibited old pleuritic adhesions and hypostatic congestion at the bases; the heart weighed $9\frac{3}{4}$ oz., and is described as normal; the liver was $31\frac{3}{4}$ oz., somewhat fatty; the kidneys were normal; the exophthalmos had quite disappeared after death; nothing, with the doubtful exception of the excess of morbid fat, was found in the orbit; the cervical chain of sympathetic ganglia was removed on the left side, but presented nothing abnormal, either to the naked eye or to microscopic examination.

CASE 3.—E. S. C—, single, æt. 23, telegraph clerk, admitted in June, 1880. Cousin insane; no inheritance of disease known. Cause unknown. Symptoms had lasted two months before admission. They began with reserve and depression,

and she became slowly more depressed and restless. She was sleepless and excited, refusing food ; she kept saying that she ought to have died, that it would have been better for every one if she had died, that I was right in saying she ought to have died. Her father thought that she listened to voices. She refused her food, saying there was something on her mind, and that she would die in a few minutes, and that it would be all the better for others if she did. She seemed depressed, but was very restless.

On admission she is described as hearing voices, and noises in her ears ; she has also experienced flashes of light. She thought people were watching her. Menstruation had been regular. She was a tall, slight girl, with large dark eyes, eyeballs slightly protruding—*exophthalmos*. She had a nervous manner, and was emotional. Complained that she had bad thoughts, and wished to die. She was first treated with shower-baths daily, and then tincture of belladonna was given internally. For the next few months she steadily gained in weight, but there was little or no change in her mental condition. It is reported that at times she was very excitable and troublesome, and also noisy. Four months after admission she was said to be slightly better in mind and body, but she soon became more excited again, and in the earlier part of 1881 she had to be removed to a lower gallery, in consequence of her violent and destructive habits. In July, 1881, she had a pulse varying from 120 to 140, eyes uniformly prominent, pupils widely dilated, and marked increase in the circumference of the neck. This patient was full of all sorts of miserable ideas—thought she was unnatural, that she was not a woman but a beast, that she had caused all the troubles and all the distresses that she saw around her in the hospital, that she must die, that she could not get better. She fully recognised where she was. She remembered her friends, and at the same time said she did not want to see them. At times she was dirty in her habits and obstinate about her food. She pinched and injured other patients if not carefully watched, but she kept her clothes on and was fairly tidy.

In this case we have only given the history so far as it had gone while she was in Bethlem, but from our experience of the other two cases, so similar in respect of age and symptoms, one feels quite sure that the end will be the same.

I do not for a moment wish to insist upon this disease having very special mental symptoms associated with it; all I would say is that the disease seems nearly associated with nervous changes, and that, as far as I have yet been able to make out, these changes are not to be found in the cervical sympathetic at all events. In all these cases there was depression at one time, followed by the most violent mania—mania of the noisy, destructive, incoherent kind, not of the simple delirious form. In all the cases there were periods of quiet and of improvement. I regret to say that I cannot attribute any of their improvement to medical treatment, for although they were all treated according to the newest lights—with belladonna, digitalis, arsenic, shower-baths, and galvanism—the results were the same. In all cases there was a tendency to impulsive violence, to refusal of food, and to dirtiness in habits, so that these patients were not only negligent, but absolutely filthy in their behaviour. In each the sexual functions seemed little or not at all affected, menstruation being recorded as regular in all three. In all these cases the patients were single.

We will now speak of some other cases, recently under notice, having some of the symptoms of exophthalmic goitre. In one case, at all events, the symptoms are recurrent and periodic, and in the other progressive, but associated with that fatal form of nervous disease—general paralysis of the insane. The first case that I report is that of a young lady who suffered from recurrent mania, and in whom most marked exophthalmos is present with each recurrence of excitement.

A. M—, single, æt. 21, governess. Over-study the supposed cause of insanity. Her mother is insane, and her father was eccentric and brutal in the extreme. The patient had suffered from headaches for years. She had scarlet fever two years before admission. The first attack of insanity occurred when she was nineteen years of age, and passed off; for this attack she was under treatment in another hospital. The present attack is described as having come on ten days before admission. It began with sleeplessness, and constant counting and multiplying aloud all day long. It was said that before she broke down the children under her charge noticed this

peculiar habit, which seemed to be growing upon her—this habit of exercising herself with figures as if to distract her attention. Her wild appearance, and her statement that she never slept, but kept awake in order to repeat tables out loud, and other odd behaviour, are recorded as signs of her insanity. She is said to have become totally changed in manner, threatened her aunt with violence, and became untidy in her appearance. She took a dislike to others of her friends, and was violent, destroying her clothes and refusing food. On admission she had delusions to the effect that her relations were against her, and also that she had a certain amount of study which must be done within a fixed time. She had not slept for two or three nights, refused food, but was easily managed. Menstruation regular. On the night after her admission she was muttering through the whole night, and was a good deal excited. She complained of headache. The next day she slept for four hours, and the attack gradually passed off. The next point of interest in the case is that although some of the attacks were synchronous with the menstrual periods, others occurred between them at irregular intervals. There was some dysmenorrhœa, but no menorrhagia. The temper and temperament of the patient were opposed to those generally seen among the hysterical. She was distinctly strong-minded, and struggled against these attacks, which she felt to be terrible in their effect on her nature and character, and to be destructive to her self-esteem. The attacks became more frequent, and left her with but few days of quiet between, till she was put under the influence of hyoscyamine, which acted most powerfully upon her, and by judicious administration prevented the recurrence of the attacks for some months. After she had been on leave of absence without medicine she again broke down, and was again under treatment for the same conditions from which she was suffering on admission, though I think the hyoscyamine did not act so readily and efficiently as it did when first given nine months before. It may be well to give a description of one of her attacks. The first symptom noticed is a staring condition of both eyeballs, with a tendency in the hair to become dry, stiff, and upraised from the head. At the same time the patient is seen to be restless, avoiding contact with her fellows, and especially avoiding recognition by the doctors.

For a few hours after the almost sudden onset of these symptoms she remains in much the same condition, if no medicine be given. She then goes to bed, but talks, counts, and repeats the whole night through, not seeming to close her eyes for a minute. Next morning she looks haggard and worn, the eyes being much more prominent, the hair much rougher, the complexion more sallow. By this time she is either slightly emotional, rushing about, or dull and heavy, seeking corners of darkness where she will not be seen by the doctors or others who may be in the ward. Her whole mode of dress and of decoration is changed. She no longer takes the slightest interest in books or in her old accomplishments. In this condition she may pass another twenty-four hours, and then, after an emotional storm and an improved appetite she slowly passes from the attack to a period of health; from the first onset of the attack to this conclusion a period of from two to five days will have elapsed. In her case the pulse at these periods has been rapid, and between the intervals it is still rather rapid, but normal as far as the tracing is concerned. There is no other disease to be noticed beyond the prominent eyeball, the prominent neck, and a pulse varying from 100 to 140.

This patient has now recovered sufficiently to go from the hospital, at first on prolonged leave of absence, and later well. She has followed a steady and rather arduous occupation, and though once or twice she was a little upset, she has kept well enough to be at large and to be considered by her employer perfectly sane.

I fear with the strong nervous inheritance and the general unstable condition of the case that sooner or later we shall see her again suffering from insanity, and then the progress of the other diseased conditions will be compared. There are several points of special interest which have been noticed in reporting this case, but which I would once more allude to—the strong inheritance, the recurrence of the insanity, associated with the recurrence of the symptoms of Graves' disease; besides this the fact that at least for a time there was marked improvement under the treatment by hyoscyamine.

The second case I would call attention to is that of E. N—,

a male, married, 39, a commercial traveller, whose disease was supposed to be due to business anxiety. No relations have been similarly affected. This is his first attack, and has lasted three months. The first symptom detected was alteration in his general manner. This alteration became more marked. He claimed persons whom he had never seen before as friends. His memory seems to be affected, and he denies having done acts which he has only just finished. His wife says that he has been writing letters and ordering goods without reason or necessity. He was full of plans for his future quite inconsistent with his position in life. He fancies he is of large means, and can spend as much money as he likes. He was at times very excitable, especially at night, when he fancied he was in other towns. He took little notice of his children, was morose with his wife, and altogether changed in temper.

On admission, he had great ideas of his property, was willing to give money away, and constantly ordering things. He believed he was possessed of large amounts of jewellery. He was a good-looking, rather slight man, eyes very prominent, pupils contracted, hair thin. He was constantly walking about, with a rather shaky, unsteady gait. There was a shakiness, too, about his hands. The knee-reflex was quite absent in the right and only slightly present in the left leg. The body was well nourished, skin slightly greasy, tongue tremulous. He was constantly in the habit of telling us that he was going to join the Horse Guards, that he would put an end to all wars, that he was going to buy large numbers of horses, and was as well as he had ever been in his life. He was restless to an extreme, so that he never seemed able to sit for more than two or three minutes at a time, but would get up, and in a purposeless way walk hither and thither in the airing courts. There is no doubt about his symptoms being markedly ataxic. On closure of the eyes the patient staggers and reels, and would fall if he did not open them again. There is no marked prominence of the thyroid body, but the pulse varies from 100 to 150, and the eyeballs are very prominent.

This last case is one of several similar ones in which marked general paralysis of the insane has been present with symptoms of Graves' disease. At present I am not prepared to say

whether the one disease occurs independently of the other or whether the two diseased processes depend upon one central nervous degeneration.

The association is further of interest from the fact that both general paralysis and Graves' disease have been considered as due to diseased states of the cervical sympathetic.

To sum up my experience, I should say that among the insane Graves' disease is more common than among the sane; that with this disease the mental symptoms are of a melancholic type. This may be readily explained as a secondary mental result, for on more than one occasion have I been able to trace the growth of a delusion of suspicion; in fact have traced the growth of an idea that a person was being watched to the circumstance that the person was peculiar in appearance, or was developing some peculiarity. Many a sane person with a physical defect becomes morbidly sensitive to his defect, and the stammerer or the man who squints is often irritable or shy.

I have seen one case in which the myxœdematous change in aspect caused a woman to become suspicious, irritable, and violent.

And the mental aspect of an insane person depends greatly on his general conditions and surroundings before the outbreak of the disease.

So I should have expected that persons of nervous stock developing Graves' disease would almost certainly be suspicious, and would fancy others were watching them, and would very likely suspect persons of influencing or affecting them. In our cases we had more than this, for the melancholia in all passed into most violent mania, hallucinations were present, and the patients presented examples of mania of the most violent, destructive, and dirty kind.

I have said there was something special in the type of insanity, and yet I have been unable to give any very distinctive characteristics in writing, for mania following melancholia is common enough under other conditions; and I can only further say that with the marked symptoms of Graves' disease, I have generally met with melancholy of the suspicious type, followed by mania of a very violent kind, with tendency to emaciation and death.

Between the fully-developed disease and insanity we have seen

some connexion, and I would call attention to the case of A. M—, for here we have conditions which have not been described, though I have seen at least one photograph of a case of recurrent mania in which the prominence of the eyeballs was very remarkable.

In this case the recurrences of insanity were always associated with recurrences of the symptoms of exophthalmic goitre, at least as far as most marked exophthalmos and rapid pulse were concerned. In this case strong neurotic inheritance is present, and I have already noticed that in the cases I have described this taint is common. This patient after a long time, nearly two years, was discharged from the hospital and has kept well.

In her case slight exophthalmos at times was present in the intervals between the attacks, associated with some emotional disturbance of a less marked nature than the fully-developed attack.

The case of general paralysis not having been watched to the end is so far unsatisfactory, and I shall hope to be able to record at some future period a complete case of that kind which may be of service both in the study of Graves' disease and of general paralysis of the insane.

CASES

OF

EMPHYEMA IN CHILDREN TREATED BY REMOVAL OF A PORTION OF RIB.

BY W. ARBUTHNOT LANE, B.S.

In empyemas, which have become chronic, portions of rib are removed, either to allow the free discharge of pus, or to permit of the approximation of the ribs and the obliteration of the cavity.

This cure I find often attributed to the removal of a portion of the bone, allowing the chest wall to fall in and approximate to the retracted lung, rather than to a more perfect system of drainage.

Peitavi, in the report of his cases, attributes the closing of the cavity to the free drainage rather than to the falling in of the ribs.

Thomas, in the 'Birmingham Med. Rev.,' 1880, describes eight cases in which portions of one or more ribs were removed. Two were after spontaneous discharge of the pus externally, two after puncturing with a trocar and insertion of a drainage tube, and four after intercostal incision.

He seems inclined to attribute the cure to the falling in of the ribs rather than to the substitution of good for imperfect

drainage. I merely quote these cases as examples of an operation which is now frequently done.

There appears to be a natural tendency in empyemas to become cured if opened, and though the falling in of the ribs may be one means, the expansion of the lungs, and the ascent of the diaphragm are probably much more important factors.

There are of course cases in which no amount of drainage would be of any use, but in many of these you would have practically to remove the whole bony framework of the side of the chest to allow the surfaces to come together. I am not going to discuss the treatment of empyemas which have ruptured spontaneously outwards, or which have been incised, but the primary treatment in the case of children.

The best method appears to be that by which the most perfect drainage is obtained.

That the usual one of incising the intercostal space fails frequently is seen often enough.

Now I would suggest that a portion of rib or ribs be removed at first, and the cavity thoroughly drained from the beginning. This treatment I was led to pursue by the observation of five cases, of which I will give a description later.

I have not succeeded in finding any account of a similar treatment.

In children the spaces between the ribs are small, and become much more so, owing to the falling in of the chest and approximation of the ribs after the cavity is opened, so that a soft tube has its calibre narrowed or obliterated, and if a firm one be used, its sectional area is necessarily small and liable to become occluded by the masses of tough, caseous, and often pultaceous lymph, which are frequently too large to come away through an intercostal incision. This material takes some time to break up, and by its presence serves as a source of irritation, preventing the approximation of the surfaces of the abscess cavity. At the same time, to prevent the tube being forced out by the movements of the adjoining ribs, its inner aperture cannot be rendered flush with the inner wall of the chest, but must be made to project some way into the space, and much pus must remain which cannot escape, except when the cavity is washed out; lotion is then substituted for pus, and there is still a foreign material separating the walls of the cavity, which prevents

healing. The tube is frequently obliquely placed with the inner end projecting upwards, owing to the shape of the rib surfaces.

A hard tube also causes much pain by its pressure, sometimes necessitating its removal.

It not infrequently causes caries of the adjoining ribs, and this is often a troublesome complication, remaining after the abscess cavity has closed.

In removing a portion of rib at the most dependent part of the cavity, an opening is obtained sufficiently large to allow of the introduction of the finger, and an india-rubber tube large enough to ensure its calibre remaining free can be fixed in, so that its inner opening shall be flush with the outer wall of the abscess. At the same time, it is not pressed on by the ribs above and below, so that there is not any chance of its being displaced.

In none of my cases had I any trouble with the growth of bone interfering with the opening except in the first one. This is often a source of much trouble in resection of ribs, as in the case reported by Dr. Taylor and Mr. Howse, '*Clin. Soc. Trans.*,' vol. xiii, where the growth of new bone was so rapid that although a large area of ribs and periosteum was removed, the opening rapidly refilled. Some surgeons lay much stress on the removal of the periosteum as well as the bone.

In empyemas at the base, the most favorable point at which a rib can be resected, appears to be the ninth in the axillary line. An empyema which is general or limited to the base does not extend much below that level, and after the evacuation of its contents the portion of the cavity that does so is rapidly filled up by the rising up of the diaphragm and its adhesion to the outer wall.

An old-standing empyema at the base may not extend below the eighth rib or interspace, as in time, after the ribs have approximated, the diaphragm is drawn further up by the contraction of the cicatricial tissue of adhesions and the fibroid changes in the lung, so that the cavity appears to heal from below upwards, rather than from above downwards, as occurs when an opening is made.

But if pus can be drawn off with the hypodermic syringe from the intercostal space below the ninth rib in axillary line, I believe the rib may be resected at that point without fear of

the opening being interfered with by the ascent of the diaphragm.

With the finger in the opening the upper surface of the diaphragm can be felt on a somewhat higher level than the external opening, and below the level of the latter is felt but a small angle between the costal and diaphragmatic surfaces. The cavity can also be thoroughly cleansed of its contents, as well as of the tough caseating lymph so often present, and any danger of the tube becoming occluded is removed. A short india-rubber tube about as thick as one's finger is fixed firmly into the opening vertically to the surface of the chest, so that its inner extremity does not project within the outer wall, so that there is perfectly free drainage, and the chest is entirely emptied of pus or lotion before the dressings are applied. Therefore there is not the same danger of carboloria.

The free use of the hypodermic syringe serves not only to indicate the surface area of the cavity, but often to obtain some idea as to its depth.

If after frequent punctures over a small area, from which alone pus is obtained, blood-stained pus is finally drawn off, the probability of its being a small circumscribed cavity is increased. This occurred in one of the cases I will describe.

In examining the cavity with the finger, it may be found that it has not been opened in its most dependent part, and another opening can then be made in a more favorable situation.

This would be more likely to occur in a circumscribed than in a general empyema.

The sooner the cavity heals up the less will be the amount of cicatricial tissue left in its wall and the danger of fibroid change in the lung diminished, and the greater the probability of the lung expanding and recovering.

If the cavity is freely drained from the bottom, it may not be necessary to wash it out a second time. The operation is simple. The area of the cavity is first defined. Should it be localised the best rib to resect appears to be that one above the lowest space in which pus is obtained by exploratory puncture, not going lower than the ninth rib in the axillary line. An incision is made along the course of the rib to be excised, taking care not to displace the skin upwards by raising the arm, otherwise

the internal opening and the skin incision will not exactly coincide, and there will be some difficulty in keeping the tube perpendicular to the surface.

The periosteum is then divided longitudinally and turned off the rib, and a piece about three quarters of an inch long is removed with cutting forceps.

The periosteum and pleura are punctured with a knife or director, and the opening dilated up.

The cavity is then thoroughly cleared of its contents, examined, and washed out. A short india-rubber tube is then fixed in so that its inner end shall not project into the cavity. Wire sutures are passed deeply through the intercostal tissues and tube, and to render its position more secure safety pins may be fixed through the wall of the tube, and attached to them pieces of round elastic surrounding the chest.

The edges of the skin are brought together.

A considerable thickness of gauze should be used, especially below the opening, and outside the bandages elastic bands should surround the chest at intervals, and should be sewn on to the dressings. These serve to prevent air getting in beneath the margins of the dressing during the movements of the body, as in coughing, &c., and by keeping up an equable pressure keep the ribs at rest, and so favour rapid cure. There appears to be no danger of caries, and the bony framework is as perfect after the child recovers as before the operation.

One often sees an empyema opened some way above its lower limit, and it is hardly to be expected that an abscess cavity with one fixed wall, namely, the ribs, should heal favorably under circumstances in which an abscess in another part of the body would not.

In this case the whole space below the opening must be filled up first, in great part by the ascent of the diaphragm and its adhesion to the chest wall, and the ribs are at the same time bound down permanently, and the lung destroyed by fibroid change. The patient then goes about with one side of the chest shrunken and disfigured for life.

The following cases occurred at the Victoria Hospital under the care of Dr. Ridge Jones and Dr. Evans, to whom I am indebted for permission to carry out this line of treatment, and to publish these notes.

CASE 1.—J. T—, æt. 5½, admitted under Dr. Evans, March 1st, 1882. The boy has had a cough for three or four weeks. Seven days ago he began to complain of sickness, bilious vomiting, and shivering, with cough and acute pain in his left side. His breathing became short, and he then had the same pain in the right side. He is a pale, unhealthy boy. Has a short, frequent dry cough, and much dyspnœa. Requires to be propped up in bed.

Right side of chest dull below the lower angle of the scapula, and above that resonance is impaired to the apex. In front, tympanitic sound in infra-clavicular region. Below that, deficient resonance, and below pectoralis major complete dullness. Above, in front and behind, loud tubular breathing. Below spine of scapula it becomes distant, but can be heard to extreme base.

Resonance at base of left lung is somewhat deficient and the respiratory murmur is harsh. Apex of heart beats in sixth space, an inch outside nipple line; temp. 102·6°. Drew off cloudy serum from the right base with hypodermic syringe.

March 2nd.—Dyspnœa more troublesome; temp. 103°. Putting an aspirating needle in at the base I only succeeded in obtaining an ounce of very turbid serum.

5th.—Temperature has fallen to normal for the first time since his admission. Sweats profusely at night. Cough very troublesome. Less dyspnœa.

9th.—Temperature runs up at night time to 100° or 101°. Right side appears rounder than the left. At lower end of sternum it measures 11 inches, left side being 10½ inches. It expands but slightly. Area of dullness has increased, and no breath sounds are heard in dull area. Spine convex to left side. Liver down to umbilicus. Some clubbing of fingers. Drew off pus from base with the syringe. The boy continued to get worse, with rapid rises and falls of temperature.

14th.—I made an incision along the seventh space in axillary line, and exposing the seventh and eighth ribs, I cut away with bone forceps the lower half of the thickness of the seventh and the upper half of the eighth rib, so as to make an opening into which I could introduce my finger. Fourteen ounces of pus came away. A large drainage tube with a central wire coil was inserted, fitting easily into the opening. It was the same

diameter as the tubes used in the other cases. Next day the boy was much easier and his temperature was normal.

On the 16th his temperature ran up to 100.8° , and I found the tube had been forced out by the movements of the approximating ribs. I then found some difficulty in replacing the tube, having finally to make it project a little way into the cavity to make sure it should not slip out again.

On the 18th he began to whoop. Later I had to use a smaller tube, owing to the contraction of the opening and the approximation of the ribs. It was retained longer in the opening than seemed necessary for the cure of the cavity, as it was feared that pus might be retained. The attack of whooping-cough was severe and completely prostrated him.

He left the hospital on the 10th of May with a dry scab over the seat of the opening.

I came to the conclusion that the removal of a portion of the thickness of adjoining ribs was not satisfactory, as the tube was liable to be displaced by the movement of these ribs, and the aperture became rapidly narrowed by the approximation of the ribs. Also that I ought to have made an opening lower down, as pus collected between the times of dressing. After examining the bodies of several children I concluded that if pus could be obtained with the needle below the ninth rib in the axillary line, that that was the most favorable rib to resect.

I saw the boy again on the 15th of October. Both sides of the chest expanded equally and well. Each measured 11 inches. No difference in resonance or respiratory murmur. Clubbing of fingers had disappeared. Spine quite straight. He had gained flesh and been very well since I last saw him. Only a slight irregularity of the margins of the ribs could be felt at the seat of excision.

CASE 2.—M. R—, æt. 2, admitted March 13th under Dr. Evans. Has been ill for many months, and much neglected. Much dyspnœa, and rapid feeble pulse. Congestion of lips and face. Moist sounds generally in left lung, especially at base. Right side dull all over. Pus drawn off with exploratory needle from lower half of chest. Tubular breathing below clavicle and above spine of scapula. Below that very distant tubular breathing is heard.

I removed three quarters of an inch of the eighth rib in the axillary line, as I could not obtain pus lower than the eighth space, and let out more than ten ounces of pus. Large masses of tough caseous material were removed through the opening. The lung felt very dense and did not appear to expand at all. The cavity was washed out and a tube fixed in. The child, which appeared before the operation to be dying of its bronchitis rather than of the empyema, did not improve much afterwards. There was very slight discharge on each dressing and none retained in the cavity), and though it was washed out a second time, it was an unnecessary precaution, as the lotion returned clear. She died on the 20th of March, the eighth day after the operation.

Post-mortem.—Pus in tubes of left lung; old pleuritic adhesions in left side. Right lung densely cirrhotic and airless at base, and to a less degree at apex. Its under and outer surfaces were attached to the wall of the chest and upper surface of the diaphragm by a dense cicatricial layer. The adhesions were not so dense at upper part of lung. The area of the empyema which had been opened could be distinctly made out by tearing away the lung from the chest wall. The surface of the lung has united to the chest wall throughout, leaving only the opening for the tube, but the adhesions were broken down with the finger easily, compared to the dense adhesion around the space. The area of recent adhesion measured $3\frac{1}{4}$ inches transversely by 3 vertically, and it had only extended downwards to the eighth space, showing we were correct in our assumption. The interval below was filled up by a dense cicatricial tissue which attached the diaphragm to the chest wall.

The rapidity with which the surfaces adhered was remarkable, as the child died on the eighth day after the pus was let out. It took place under very disadvantageous circumstances, as the child was during the time very ill with bronchitis.

CASE 3.—E. S—, æt. $4\frac{1}{2}$, admitted June 12th, under Dr. Evans. Was never a strong child. Had whooping from Christmas till quite recently. A fortnight ago she caught cold and had fever, cough, with acute pain in the left side. At first dyspnœa was severe, but now she is more comfortable in that

respect. Has lost flesh rapidly. Some diarrhœa. Profuse night sweats.

On admission.—Some dyspnœa; resp. 40, pulse 122, temp. 100°. Left side expands but slightly. Heart displaced to right side, the apex beating below xiphoid cartilage. Dulness all over left side anteriorly and posteriorly, also absence of respiratory sounds, except tubular breathing, at extreme apex. Puerile respiration over right lung. Exploratory punctures showed presence of pus at different levels.

June 15th.—Eighteen ounces of pus drawn off. She improved slightly, but soon the breathing again became rapid, the night sweats profuse, and her appetite bad, and she became very much wasted.

30th.—Apex of heart one inch below and to inner side of nipple. The front of the chest is much clearer, but behind it is dull. Friction sound and distant tubular breathing heard posteriorly.

July 3rd.—On using a small aspirating needle, I could only obtain pus from a small area of eight or nine square inches between the scapula and spine. After puncturing this area frequently, I drew off blood-stained pus.

I cut down on the lowest rib, viz. the eighth, below which pus was obtained, and examining the interspace, found that had I intended to put in a drainage-tube, owing to the very small interval between the ribs at this point, it would be almost impossible to do so.

I then removed three quarters of an inch of the eighth rib, two inches and a quarter from the median line. About three or four ounces of blood-stained pus came away. Even after the removal of a piece of rib, the opening was a comparatively small one, and I was obliged to use a smaller tube than usual.

After the operation the child rapidly improved. At first the night sweats diminished, and then disappeared. The discharge from the wound was very slight. Till the tube was removed on 26th July, her temperature did not rise above a degree.

She went to the seaside early in August with the wound quite healed.

The air then entered the lung freely, and there was but little difference between the percussion notes of either base.

In this case the empyema was at first general or nearly so,

as, before tapping, the exploratory needle showed the presence of pus up to a fairly high level, I think up to the third space, but of this I will not be certain, not having made a note of it at the time.

After tapping it became localised to a small area at the back of the lung.

This partial obliteration of the cavity appears frequently to take place after tapping, and might suggest the advantage of first tapping, and later opening the residual cavity.

I saw the child on 15th October, and found a slight irregularity at the point of resection. Otherwise the rib was perfect as before.

Both sides of the chest expanded well and equally. No difference in resonance or respiratory sounds, or in measurement of either side. Apex of heart in normal position.

CASE 4.—H. B—, æt. 4, admitted July 11th, under Dr. Ridge Jones. Whooping-cough at Christmas. Severe bronchitis during the last two winters. For the last seven weeks has had a bad cough, with much shortness of breath and profuse night sweats.

On admission.—Much dyspnœa and restlessness. Left side is dull almost up to the clavicle. Breath sounds cannot be heard in lower part of chest, but in the upper part tubular breathing is heard in front and behind. The side moves but little. Right side appears to be quite healthy.

Heart's impulse diffused over a large area, and area of pre-cordial dulness extends a little to right of sternum. Heart sounds clear. Pulse rapid and feeble. Spleen felt below the ribs.

About twenty ounces of pus drawn off with aspirator. For some short time after the operation he appeared to be easier. The dyspnœa and sweating came on next day, and he had to be propped up in bed.

July 13th.—Finding by the exploratory needle that pus had again collected in the lower half of the chest, and obtaining it in the ninth space, I removed three quarters of an inch of the ninth rib in the axillary line. About fourteen ounces of very thick pus came away.

He breathed so badly during the operation, that after letting

out the pus and washing out the cavity I fixed the tube in as soon as possible, and did not examine the cavity digitally.

The dyspnœa remained, and there was little perceptible improvement in the general condition of the patient. At times, especially at night, he had severe attacks of dyspnœa, followed by profuse sweats, after which he became more comfortable.

As there appeared to be pus somewhere, the exploratory needle was passed into many of the spaces from the apex to the base, and no pus was obtained.

Toward the base, it passed through firm resisting tissue. The cardiac area was not punctured, the presence of pus in the pericardium not being suspected. The large area of cardiac impulse and dulness was attributed to the right ventricle being enlarged from pulmonary obstruction, due to chronic bronchitis, &c. The cavity of the empyema was shallow, and a probe could only be passed into it for an inch, and it only retained a few drachms of carbolic lotion.

The boy then got worse, and died on 5th August, twenty-two days after the operation.

Post-mortem.—Upper lobe of left lung tolerably free from adhesion. The under surface of the base and outer surface of the rest of the lung was united to the diaphragm and wall of the chest by dense layer of fibrous tissue. Only a small cavity, $1\frac{1}{2}$ inches by $1\frac{1}{2}$, remained unhealed; above, the inner aspect in front was formed by a firm wall formed by condensed lung and pericardium, forming together a layer nearly half an inch thick, and below by pericardium alone. The cavity was in free communication with the opening and was apparently closing up rapidly.

With the exception of the upper lobe the lung was completely carnified and airless. Little change had taken place in the ends of the rib, which appeared healthy. The pericardium was much thickened and enormously distended with pus, and the heart covered by a thick layer of sticky pus.

Except for some adhesions, the right lung was healthy. The result made one regret that at the time of the operation the child was too ill to allow an opportunity for careful digital examination of the cavity, as the bulging pericardium might then have been detected and drained.

CASE 5.—J. M—, æt. 3, admitted July 22, 1882, under Dr. Evans. Caught cold five weeks ago, and had inflammation of right lung and pleurisy. Has not been well since that date, being confined to bed. Has lost flesh considerably. Night sweats.

On admission.—Right side completely dull, rounded, and bulging. Pus can be drawn off at any level below the clavicle. No breath sounds to be heard. Heart considerably displaced to the left side, and the liver well down.

I removed three quarters of an inch of the ninth rib in axillary line, and a large quantity of pus came away, but as at each expiratory effort it was driven in a jet for several feet it could not be measured. A tough caseating mass of semi-organised lymph, forming a cast of the angle between the ribs and the diaphragm, was removed through the opening.

The finger could be passed generally through the cavity and the surface of the lung was felt to be sticky and free from adhesion. The lung felt soft and elastic.

The finger rested on the diaphragm, and there only remained a small interval between the ribs and diaphragm below the level of the opening.

This case did remarkably well. There was very little discharge at each dressing, and the cavity rapidly filled up. On the 30th August, as it appeared to be quite well, the tube was removed, and the child left the hospital on the 11th September quite well.

I saw the boy on the 15th October. He had been quite well since his return home, and was much stouter.

The ends of the rib were united by a circular flat plate of bone, and it was not adherent to the rib above or below.

There was no difference in the measurement, resonance, or respiratory sounds on the two sides.

ABNORMALITIES

OBSERVED IN THE

DISSECTING ROOM OF GUY'S HOSPITAL

DURING THE

SESSIONS 1880-81 AND 1881-82.

By R. E. CARRINGTON, M.D., P. HORROCKS, M.D.,

AND

W. HALE WHITE, M.D.

DURING the two sessions of 1880-81 and 1881-82, the subjects received at Guy's Hospital numbered 183. Of these one or two were used for operations on the dead subject, but the rest were dissected by the students, who were requested to call the attention of the demonstrators of anatomy to any abnormality met with. But doubtless, in several instances, unusual structures or relations were unnoticed, or attention was not called to the part, so that the following record does not profess to give anything but an approximate idea of the relative frequency with which the abnormalities described are to be found in various subjects.

BONES.

No abnormalities in the osseous system were recorded.

MUSCLES.

In one subject a distinct muscular slip, about half an inch wide, with no tendon at either end, extended from the fascia covering the serratus posticus superior to the superior curved line of the occipital bone, just external to the protuberance. This muscle was covered by the trapezius, lay on the splenius, and was present on the left side. Muscular slips somewhat like this are described in previous volumes of the 'Guy's Reports.' In one case,¹ a muscular slip extended from the mastoid process to the upper border of the serratus posticus superior, and in another,² a tendinous slip arose from the first dorsal spine, ran over the serratus posticus superior, to be inserted with the lowest slip of the splenius into the posterior tubercle of the third cervical vertebra.

Three cases occurred in which an abnormal muscle was placed transversely in the upper part of the neck, below the ear. In the first case a delicate muscle was seen stretching from the occipital bone just below the protuberance to the fascia over the masseter; it was a quarter of an inch long and had a tendinous, bony attachment, and also a tendinous intersection at the posterior third of the sterno-mastoid. The second case was on the opposite side of the same body, whilst the third was slightly different; thus it arose by two little tendons from just below the occipital protuberance. Each soon joined a fleshy belly, and the two bellies united to form one muscular slip which was inserted into the skin half an inch below the lobule of the ear; this muscle was a quarter of an inch wide at its widest part, and was present on the right side only.

The muscle corresponds in origin to the "transversus nuchæ" described by Macalister,³ but its insertion is rather different from any of those he mentions, which were inserted either into the outer part of the superior curved line of the occipital bone or the posterior border of the sterno-cleido-mastoid, or blended with the retrahens aurem. Schultze met

¹ 'Guy's Hosp. Reports,' 3rd Series, vol. xvi, 1871.

² Ibid., 1869.

³ 'Proceedings of Royal Irish Academy.'

with this muscle in eighteen out of twenty-five subjects, but Macalister found it in only seven out of twenty subjects. The experience at Guy's would lend support to Macalister's statement, but it must be noted that students not unfrequently cut away small muscular slips without calling attention to them. Only the other day we accidentally saw a student removing a good example of the transversus nuchæ with the deep cervical fascia under which it lay. The muscle has always been symmetrical. Knott has also recorded two examples of this muscle in the '*Journal of Anatomy and Physiology*,' 1881.

In one subject there was a thick muscular slip half an inch in width, which arose in common with the lower part of the levator anguli scapulæ, and went quite distinct from that muscle to be inserted into the fascia covering that side of the serratus magnus which is next to the thoracic wall.

According to Walsham¹ the omo-hyoid has a clavicular origin once in every twenty subjects; only one instance was found in our dissecting room. This is such a common abnormality that, in addition to the numerous instances given by Wood, many examples are to be found in the '*St. Bartholomew's*,' '*St. Thomas's*,' and '*Guy's Reports*.'

In another case an abnormal muscle in the left axilla (*coracobrachialis brevis*) arose from the base of the coracoid process, and from the fascia which lies over the subscapularis, between this and the head of the humerus; it was about an inch and a half wide, and passed downwards, curving over the subscapularis and teres major muscles, to be inserted by tendinous fibres into the upper border of the tendon of the latissimus dorsi. The muscle passed under the axillary vessels and the outer and inner cords of the brachial plexus, but over the posterior cord, more especially lying on the circumflex and musculo-spiral nerves. Similar abnormalities have been described by Wood ('*Journal of Anat.*,' vol. i) and by Macalister (op. cit.).

A third head to the biceps coming off from the humerus at the upper and inner part of the brachialis anticus was observed, but not so often as once in every eight or nine bodies as stated by Theile.

There were many instances of splitting of the extensor

¹ '*St. Bartholomew's Hosp. Reports*,' 1880.

communis digitorum tendons in an unusual way ; and some of them were very complicated.

On the right side one tendon divided into two for the index and middle fingers. The ring finger had two muscular slips from the extensor and two tendons, and the little finger had two tendons. All these were parts of the extensor communis digitorum. Various arrangements of these tendons are common, but in the *résumé* by Prof. Humphrey¹ of them, there is none exactly like this. In another example, a muscular slip from the extensor indicis, divided anteriorly into three parts, one of which spread out over the second dorsal interosseous muscle, a second spread over the anterior part of the back of the third metacarpal bone, and a third spread out into the fascia covering the third dorsal interosseous ; also the extensor minimi digiti gave two slips to the extensor communis digitorum. Once an extensor ossis metacarpi pollicis longus was noted ; it arose from the lower third of the external intermuscular septum, between the extensor carpi radialis longior and supinator longus ; at the junction of the upper and middle third of the forearm it ended in a long tendon which was inserted into the base of the metacarpal bone of the thumb. This seems to be a very unusual muscle. Quain makes no mention of it.

A supinator brevis accessorius was present on both sides of one subject. On the right side it arose by two bellies from the humerus, one coming from just below and behind the insertion of the deltoid and from the fascia covering that muscle ; the other from the outer surface of the humerus for three inches above the elbow, thus encroaching considerably on the origin of the brachialis anticus, and from an intermuscular septum between the brachialis anticus and the supinator longus. The second belly was broad and flattened from before backwards. These two united and mainly ended in a pointed and somewhat flattened tendon, which was inserted into the oblique line of the radius just below the tubercle. From the belly which arose lowest down another slip was given off, which was at first muscular, but finally became tendinous, and was lost on the fascia covering the deep muscles of the forearm. The muscle was considerably concealed, being overlapped by the

¹ ' Brit. Med. Journ.,' vol. ii, 1872, p. 53.

biceps, and lying between the supinator longus and brachialis anticus. On the left side, the upper origin was absent; the muscle was consequently smaller, but otherwise it was the same as on the right side, and had two insertions. This rare muscle is described by Henle.

In another subject was an accessory abductor minimi digiti manûs, which arose by two fleshy slips from the deep fascia of the forearm over the tendons of the flexor sublimis digitorum. Its upper head was long and narrow, and about two inches and a half above the annular ligament, the lower head broader and only half an inch from the annular ligament; the two heads joined to form one muscle, which passed over the ulnar vessels and nerve, on a level with the annular ligament, to blend with the abductor minimi digiti at its insertion.

Macalister regards this long head of the abductor minimi digiti as being a derivative of the palmaris longus.

In the subject described as having a double origin to the omo-hyoid, there were two pyriformes muscles on both sides. On the right side they were nearly of the same size, and arose together from the front of the sacrum and came out of the great sacro-sciatic notch. The more superficial, which had the longest tendon and was also the lower of the two, was directed across the gluteal region to be inserted into the upper margin of the great trochanter. The great sciatic nerve was split, one piece going above and the other below this muscle. The second one was deeper and was inserted into the digital fossa with the obturator internus.

On the left side the lower of the two pyriformes muscles soon joined the upper, becoming blended with it about half an inch before its insertion, which was normal.

One instance has been previously recorded in the reports¹ of divided pyriformis, but it does not seem to have been so complete as this, and occurred on one side only; but it is by no means a rare abnormality, Humphrey mentioning several cases in some of which the sciatic nerve was divided as here.²

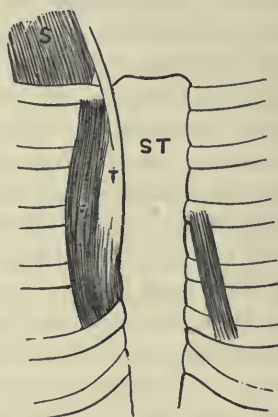
On the right side of the sternum in one case (st, Fig. 1) was a large, well-marked muscle, extending from the tendon of the

¹ 'Guy's Hosp. Reports,' 3rd Series, vol. xviii, 1873.

² 'Brit. Med. Journ.,' vol. ii, 1873, p. 78.

right sterno-mastoid (s), with which it was partially continuous, down to the cartilage of the right fifth rib. It was also attached above to the clavicle and second costal cartilage and the adjacent sternum.

FIG. 1.



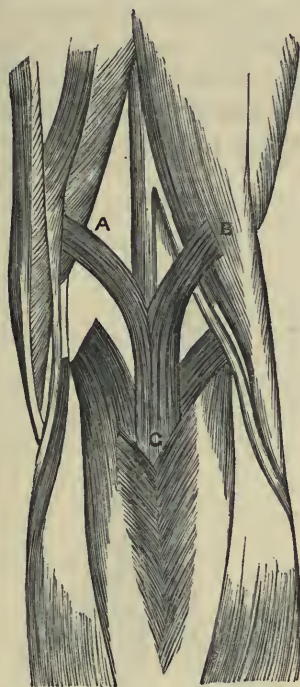
On the inner side (†) it was tendinous as low as the fourth costal cartilage. On the outer side it was fleshy all the way down. It was inserted below chiefly into the cartilage of the right fifth rib and partially into that of the fourth rib and the fascia over the pectoralis major, which was partially concealed by the muscle. On the left side was a small abnormal muscle, consisting of a few fleshy fibres extending from the left third to the left fifth costal cartilages.

Two or three other specimens of this abnormal muscle were recorded. In each the upper end blended, at least partially, with the sternal origin of the sterno-cleido-mastoid, and the lower end was attached to the costal cartilages, generally from the third to the fifth.

Fig. 2 shows an abnormal muscle consisting of a small fleshy slip, three inches in length, over the right popliteal space. It had no osseous attachments but was connected with fascia above and below. Above there were two slips, which embraced the great sciatic nerve; the inner slip (A) was connected with the fascia between the semi-membranosus and the semi-tendinosus, the outer slip (B) seemed to end in the fatty tissue ex-

teral to the great sciatic nerve. The lower part of the muscle (c) ended in the fascia over the gastrocnemius.

FIG. 2.



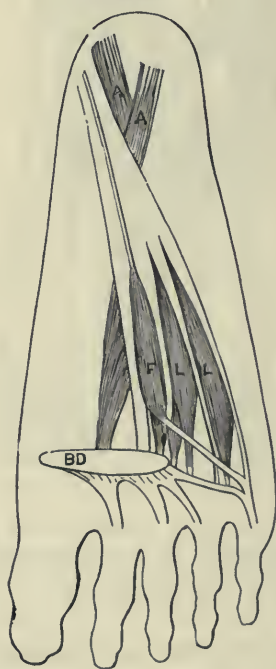
No muscle corresponding exactly with this has yet been recorded so far as we know. It was impossible to say whether it was a slip from any of the hamstrings or a third head to the gastrocnemius.

On the front of the leg the peroneus tertius was once seen to send a slip to the metatarsal bone of the fourth toe, and in the same subject the peroneus brevis sent a slip to the front of the second phalanx of the little toe. One case was noted of a very unusual fourth peroneus muscle; its origin was blended with that of the peroneus longus, from which it was separated by an intermuscular septum. It arose from this and the fascia covering it and ended in a long tendon which was inserted into the external annular ligament. There is only one other

example¹ of a fourth peroneus muscle in the Reports of the Metropolitan hospitals, and that was a commoner variety, being inserted into the os calcis.

In Fig. 3 is shown an abnormal muscle in the sole of the foot, which took origin from the tendon of the flexor longus digitorum of the right side near the heel. It arose as a slender tendon which passed forwards and outwards, becoming fleshy (F) where the flexor longus digitorum split into its tendons; the fleshy belly lay between the tendons of the second and third

FIG.



toes. Anteriorly it became tendinous again and joined the slip of tendon from the flexor brevis digitorum (BD) to the little toe. The second lumbrical muscle was absent. The third lumbrical (L) was very pale but had a distinct tendon. The first and fourth lumbricals were normal. A A represent the two heads of the Accessorius.

¹ 'St. Thomas's Hosp. Reports,' vol. vi.

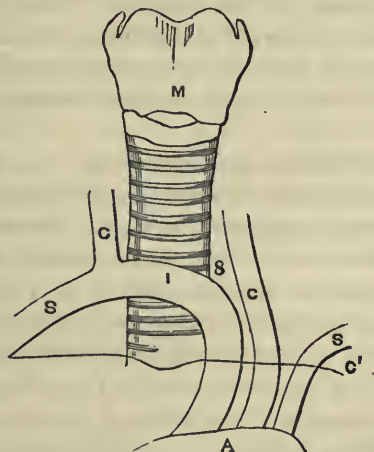
On the left side was a similar slip arising from the tendon of the flexor longus digitorum ; its fleshy belly lay on the tendon of the little toe. Just before becoming fleshy it was joined by another slender tendinous slip arising from the external inter-muscular septum. At the head of the fifth metatarsal bone the fleshy belly became tendinous and divided into two parts, one being inserted into the base of the first phalanx of the little toe, and the other winding round the outer border of the tendon of the flexor longus digitorum so as to get beneath it, where it split into two parts which were inserted into the sides of the second phalanx of the little toe. There was no slip from the flexor brevis digitorum to the little toe.

Muscular slips in connection with the long and short flexors in the sole of the foot are not very rare. The above has been recorded because it presents attachments differing somewhat from those previously recorded. This mode of formation of the flexor tendon to the little toe is, however, mentioned by Macalister (*op. cit.*).

ARTERIES.

In Fig. 4 is shown the innominate artery in front of the

FIG. 4.



trachea in the neck. This artery came off from the arch of the

aorta on the left side of the trachea, and passed up on the left side, gradually curving towards the right, so that it got in front of the trachea about an inch above the sternum. On the right side of the trachea it divided into right common carotid (c) and right subclavian arteries (s). The top of the artery as it crossed the trachea was on a level with the eighth ring of cartilage (8) counting from above. The common carotid (c) and the subclavian (s) vessels on the left side were normal except that they were perhaps a little more to the left, owing to the position of the innominate. c' is the clavicle, where it crossed the vessels. This abnormality is unique so far as we know.

It is of considerable surgical interest especially in reference to the operation of tracheotomy.

In the subject already described as having abnormal pyriformes and omo-hyoid muscles, the following arterial abnormalities occurred :

On the right side the inferior thyroid arose alone from the subclavian to the usual position of the thyroid axis and gave off no ascending cervical branch. The superior intercostal, transverse cervical, and suprascapular arose from a common axis on the second portion of the subclavian ; from this axis there arose a very large ascending cervical going up between the scalenus anticus and rectus capitis anticus major ; also from this axis a number of small branches took their origin, which ascending were lost in the muscles. There was no middle meningeal from the internal maxillary on this side, but a large branch was given off from the ophthalmic just before it entered the orbit, which running back took on the supply of the absent vessel.

On the left side the internal mammary was a branch of the thyroid axis, which was otherwise quite normal ; the inferior thyroid had a distinct ascending cervical. There was no superior intercostal. There was a very large branch from the second part of the subclavian, which ascended on the middle scalene muscle, lying on the posterior tubercles of the seventh and sixth cervical vertebræ ; it then turned backwards over the latter at a right angle, passing therefore between the posterior tubercles of the transverse processes of the fifth and sixth cervical vertebræ ; it then continued up behind the posterior tubercles of the transverse processes of the remaining cervical vertebræ and anasto-

mosed with the occipital. The posterior scapular was a branch of the third part of the subclavian.

In a previous volume¹ of the 'Reports,' a case is mentioned in which the profunda cervicis took somewhat the course it did here, coming, however, from the first instead of the second part of the subclavian, but it turned back between the fifth and sixth transverse processes as in this case. In another volume² many abnormalities are given as happening in the branches of the third part of the subclavian, but none of them are so complicated as in this subject, which is remarkable for having muscular abnormalities also.

In another subject the only representative of the thyroid axis was a small inferior thyroid which after giving off the ascending cervical ended in the sterno-hyoid and sterno-thyroid muscles. The transversalis colli and suprascapular both came off from the third part of the subclavian, and the lower part of the thyroid gland was supplied by a branch of the common carotid which arose one inch above the termination of the innominate; it was as large as a normal radial and passed transversely inwards at right angles to the carotid. This is one of the rarest branches of the common carotid.³ Another arrangement noticed once only was for the vertebral and internal mammary to come off together and the inferior thyroid to be a branch of the latter.

In several cases two or more branches of the axillary artery came off from a common axis just as the transverse cervical, &c. come off from the thyroid axis. Two of the most striking of these were as follows:—one in which the axillary artery gave off a large branch on its inner side just below the acromio-thoracic; this passed under the vein, and gave origin to the following, all arising together—the long thoracic, the external mammary, the subscapular, the dorsalis scapulæ and the posterior circumflex: and another in which there was an axis from the second part of the artery giving off the superior, long, and acromio-thoracic arteries; and another axis from the third part giving rise to the dorsalis scapulæ, subscapular, and posterior circumflex. The posterior circumflex was occasionally observed to be a branch of the subscapular; in one instance the superior profunda came off from

¹ 'Guy's Hosp. Reports,' 3rd Series, vol. xvi, 1871.

² 'Guy's Hosp. Reports,' 3rd Series, vol. xiv, 1869.

³ 'Quain's Anatomy,' vol. i, p. 360. 8th edition.

the former artery. The only point to be noticed about the two instances just given of grouping of the axillary branches is that, in all the cases recorded in previous volumes of the *Guy's* and *St. Bartholomew's 'Reports,'* the superior profunda was a branch of the bunch of arteries, whilst, in the cases now recorded, the nearest approach to that was the solitary example of the superior profunda being a branch of the posterior circumflex. In the *'Guy's Hospital Reports'* for 1869, this is stated to be the most common axillary abnormality. Several instances occurred of a vas aberrans, and once the very rare¹ case of the anterior interosseous being a branch of the axillary; once also the ulnar came from that vessel.

An abnormal palmar arch in the right hand of one subject was formed as follows :

The ulnar artery divided into superficial and deep, the superficial offset supplied both sides of the little and ring fingers and the ulnar side of the middle finger. The deep offset joined the radial. The radial gave off a branch which divided into princeps pollicis, and radialis indicis. The deep arch formed by the radial artery and the deep branch of the ulnar gave off a large branch lying in the interosseous space, which bifurcated anteriorly to supply the contiguous sides of the middle and index fingers.

Just after giving off the interosseous trunk in the usual place, the ulnar artery gave off a long slender branch, which ran down into the palm of the hand, beneath the annular ligament, but superficial to the median nerve; it terminated by dividing into two, one of which joined the branch of the princeps pollicis to the ulnar side of the thumb, and the other joined the artery from the deep arch lying in the second interosseous space.

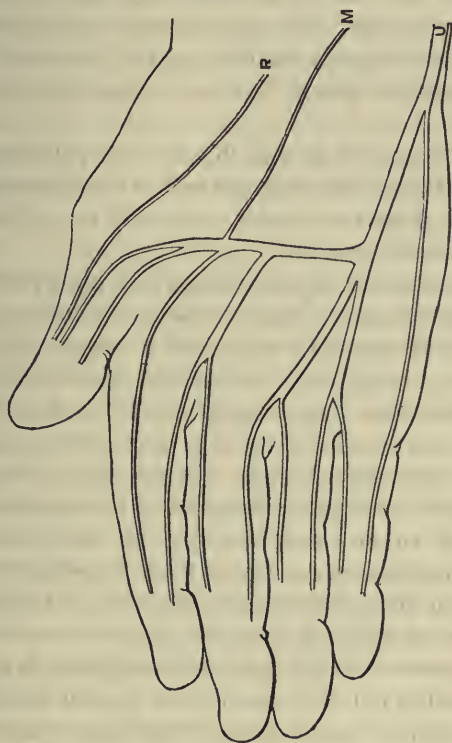
This abnormality is not infrequent and has been before recorded in the *'Guy's Hospital Reports'* (vol. xvi). See also *'Quain's Anatomy,'* vol. i.

In one case the following very unusual superficial palmar arch occurred (Fig. 5). The ulnar divided into two soon after passing the wrist; of these the more internal supplied the ulnar side of the little finger, and the other branch soon subdivided into three, of which two supplied half the little, the whole of the ring, and half the middle fingers, as shown in the figure, whilst the

¹ 1 in 506. *'Quain's Anatomy,'* vol. i, p. 398. 8th edition.

third branch went on to join the comes nervi mediani, thus completing a superficial palmar arch; from this junction one artery was given off to supply half of each of the ring and index fingers and another the other half of the index finger and both sides of the thumb, the outer side of which was further supplied by a branch of the radial which became superficial in the palm and ran forwards under the skin. The comes nervi mediani was very small. The superficialis volæ was present but ended

FIG. 5.



before joining the arch, although from its direction it looked as though the use of a fine injecting fluid would have shown it to have a connexion with the branch of the ulnar and comes nervi mediani at their junction. The only instance that we have read of at all resembling this is one in which the comes nervi mediani took on the supply of the ulnar in the palm.¹

¹ 'Guy's Hosp. Reports,' 3rd Series, vol. xvi, 1871.

In another subject an artery as large as the radial issued from the chest immediately below the third costal cartilage, and a quarter of an inch from the edge of the sternum. It was a branch of the internal mammary, passed directly outwards in the superficial fascia and, hardly at all diminished in size, entered the mamma of the corresponding side, anastomosing with branches from the axillary and intercostals. It was present on both sides.

The subject was that of a woman apparently between forty and fifty years of age. The mammæ were not larger than normal. Just below the left clavicle, near its acromial end, was an enlarged lymphatic gland, but no cause for this was discoverable.

It is not unfrequent to find the anterior cutaneous branches in the upper two or three spaces rather large, especially during lactation, but these two vessels were much larger than anything yet recorded in this region.

A most remarkable hepatic artery was observed in one case. The hepatic artery, after giving off the gastro-duodenalis superior, split into two divisions, an upper and a lower; the latter gave off the pyloric branch and then divided into two branches, each of which entered the transverse fissure of the liver; the former also divided into two, each division again bifurcating before it entered the transverse fissure. It will thus be seen that the liver ultimately received six divisions of the hepatic artery.

In another subject the liver got an additional artery by means of a large branch coming off from the pyloric branch of the hepatic, and in place of the pancreatico-duodenalis inferior there was a large vessel which, joining the gastric branch of the gastro-duodenalis, went on as a large gastro-epiploica dextra.

In one case the pyloric branch of the hepatic artery was given off in the liver and passed down in the lesser omentum to the pyloric end of the stomach.

The right kidney in one case received a special branch from the lower part of the aorta. It entered the lower end of the kidney near its inner border. A vein of similar size emerged from its upper end and entered the vena cava inferior. The normal renal artery entered the kidney with its vein, but was in front of the latter near the hilum.

Another case occurred almost identical with the above.

There were two other cases in which the vena cava received a special branch from the upper extremity of the kidney, and the aorta gave a special branch to the lower extremity: the usual renal vessels were present, but the artery lay in front of the vein.

In another subject the right kidney received two renal arteries both from the aorta, one given off opposite the left renal, and the other a quarter of an inch higher. Both vessels gave large branches to the right supra-renal capsule.

On the right side, in another subject, an accessory renal artery arose from the aorta, half an inch below the normal branch. It entered the lower part of the internal border of the right kidney, and in its course to this point gave off a spermatic branch equal in size to another which arose from the aorta above the normal renal. Both spermatics ran together and anastomosed with the lumbar arteries.

There was one case in which the kidney on the right side received two renal arteries given off from the aorta at a quarter of an inch apart: one was on the level of the artery on the left side, the other just above it; both gave good-sized branches to the right supra-renal body.

This is a common abnormality; examples of it are recorded in previous volumes of the 'Reports.'

The branches of the internal iliac were very frequently not in the position which the text-books describe them as occupying, but the varieties were more numerous than can well be described in the present short paper. Among the most common exceptions were the giving off of the sciatic from the posterior trunk together with the gluteal, and the giving off of the ilio-lumbar high up before the artery divided into anterior and posterior trunks.

In two subjects an abnormal obturator artery was given off by the external iliac artery. They took the same course as when given off by the deep epigastric, and ran down on the inner side of the femoral ring. This abnormality is much more rare than an obturator given off by the deep epigastric, of which several instances also occurred, the artery generally passing down on the inner side of the femoral ring.

A superficial plantar arch (Fig. 6) was formed as follows: the posterior tibial artery divided normally into _{the} internal and

external plantar ; the superficial branch of the external plantar appeared at the outer border of the foot between the flexor brevis digitorum and the abductor minimi digiti, and gave off

FIG. 6.



a small artery to the outer side of the little toe. The internal plantar ran forward between the flexor brevis digitorum and

abductor pollicis, and formed a very perfect superficial plantar arch, crossing the tendons of the flexor brevis digitorum and communicating externally with the superficial branch of the external plantar. The branches of the arch were four in number, each rather small. They ran in the first, second, third, and fourth spaces respectively, and bifurcated at the clefts of the toes to form digital arteries, for the contiguous sides of all the toes. The branch supplying contiguous sides of the great and second toes did not communicate with any branch from the deep arch.

The latter was formed in the usual way, but was smaller than usual. It gave off three branches lying on the interossei muscles, one in the first space joined the superficial digital between the second and third toes, one in the third space also joined the same superficial digital, and one in the fourth space joined the superficial digital between the third and fourth toes.

It was doubtful which should be called the digital arteries, for the branches from both arteries were nearly the same size, those from the superficial being perhaps rather smaller.

The digital artery along the inner side of the great toe was given off by the communicating branch of the dorsalis pedis.

Exactly the same condition was found on both sides.

In another subject the internal plantar artery sent a deep branch to join the communicating branch from the dorsalis pedis, from which were given off the usual digital offsets to the inner side of the great toe and the contiguous sides of the great and second toes.

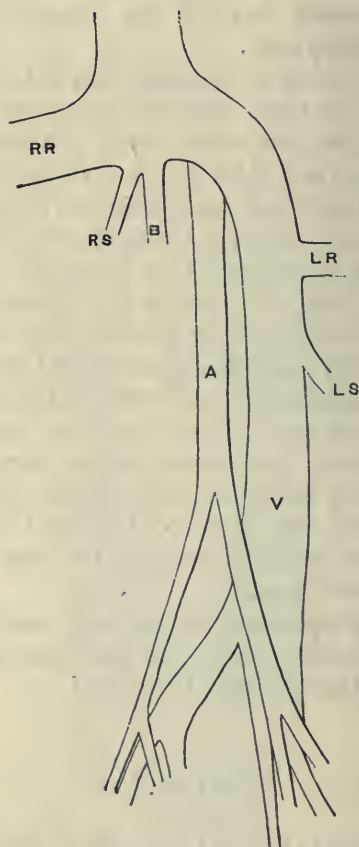
These abnormal plantar arches are much rarer than variations in the palmar arches. They are the first that have been recorded in the 'Guy's Hospital Reports.'

VEINS.

The only recorded abnormal vein was a vena cava inferior, which began on the left side of the fifth lumbar vertebra. It then passed up, slightly overlapping the left sides of the bodies of the vertebræ as far as the first lumbar, receiving on its way the left spermatic and left renal veins. It then turned across the first lumbar vertebra lying on the aorta, and though

already large it suddenly became much larger, receiving altogether a large right renal, a right spermatic, and a vein returning blood from the lumbar vertebræ. This transposition of the vein of course altered the relations of the iliac arteries and veins, as will be seen on reference to Fig. 7.

FIG. 7.



A. Aorta. V. Vena cava inferior. RR. Right renal vein. LR. Left renal vein. RS. Right spermatic vein. LS. Left spermatic vein. B. Azygos vein.

This is a rare abnormality, no other case of it being recorded in the 'Guy's Hospital Reports,' but one is to be found in the

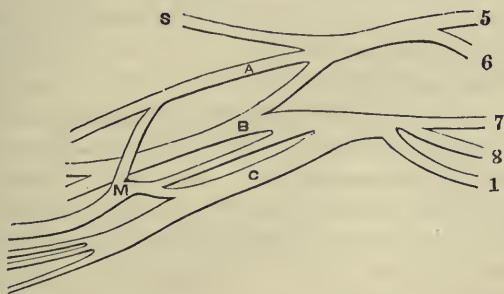
'St. Thomas's Hospital Reports.'¹ Quain mentions it as occurring occasionally, and suggests that it may be due to a persistent left cardinal vein of the foetus, the right one having become obliterated.

NERVES.

In the neck two abnormalities of nerves were recorded, one in which the vagus gave off a large branch to join the descendens noni about half an inch below its origin from the ninth, but above its union with the communicans; and a second in which the hypoglossal, just below the posterior belly of the digastric near the tendon, gave off a small nerve which entered the substance of the stylo-hyoid muscle.

There were many brachial plexuses which differed both from that described by Mr. Lucas in a previous volume of the 'Reports,' and also from that usually figured in the text-books; but the opinion expressed by Mr. Lucas, that the plexus as described by him was more common than any other variety, was fully supported by the cases we observed. It was more frequent for the abnormality to be present on both sides than on one only. Some examples are figured, and explain themselves (*vide* Figs. 8, 9 and 10),² and among those not figured

FIG. 8.

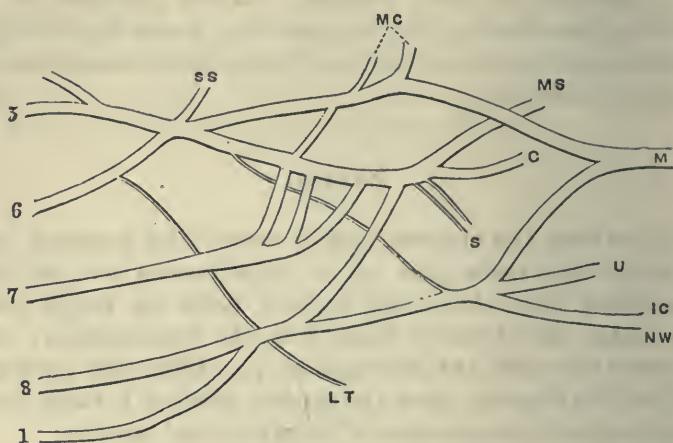


A. Outer cord. B. Posterior cord. C. Inner cord. M. Median nerve. S. Suprascapular. 5, 6, 7, 8. Cervical nerves. 1. First dorsal nerve.

¹ 'St. Thomas's Hosp. Reports,' vol. vi.

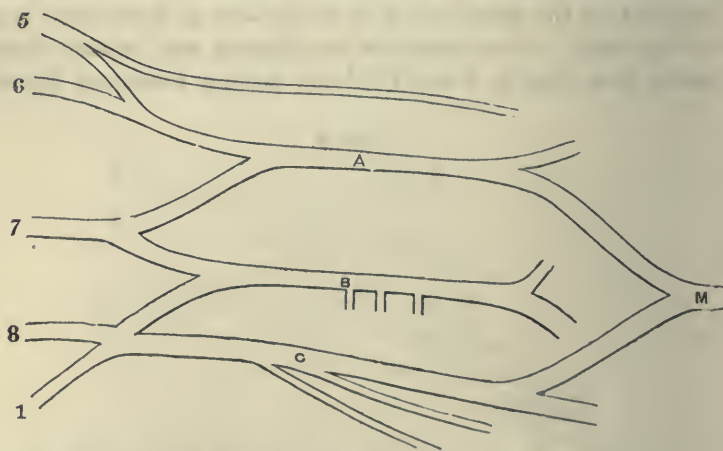
² Fig. 8 is reversed in the diagram. It should be the left side.

FIG. 9.



M. Median nerve. U. Ulnar nerve. IC. Internal cutaneous nerve
NW. Nerve of Wrisberg. MS. Musculo-spiral. C. Circumflex. MC.
Musculo-cutaneous. S. Subscapular. SS. Suprascapular. LT. Long
thoracic. 5, 6, 7, 8. Cervical nerves. 1. First dorsal nerve.

FIG. 10.



A. Outer cord. B. Posterior cord. C. Inner cord. M. Median
nerve. S. Suprascapular. 5, 6, 7, 8. Cervical nerves. 1. First dorsal
nerve.

one of the most common variations was for the median to have three heads, the additional head coming from either the outer or the inner cord; and another was for the seventh and eighth cervical and first dorsal to form a single cord. In one case the sixth, seventh, and eighth cervical each bifurcated, the sixth contributing to the outer and posterior cords, the seventh to the same two, and the eighth to the inner and posterior. There was a very complicated plexus in which there were two long thoracic nerves from the fifth and one from the sixth; the external anterior thoracic came from the sixth and seventh by two heads; the fifth had many communicating slips with the sixth; the outer cord was formed by the fifth, sixth, and seventh and the posterior by the sixth, seventh, and eighth; and the ulnar had two heads, one from the outer and one from the inner cord. This also occurred in a case in which there was no other abnormality. In another subject there were two posterior cords, one of which, derived from the fifth and sixth, gave off the short subscapular nerves and the circumflex, and the other from the seventh and eighth, the musculospiral and long subscapular; the outer head of the median came directly from the seventh and the long thoracic from the sixth and seventh. The musculo-cutaneous was occasionally observed not to perforate the coraco-brachialis, an abnormality recorded as occurring in previous years in our dissecting room.

A remarkable intercosto-humeral plexus is shown in Fig. 11. This plexus was made up of the nerve of Wrisberg (w) and the posterior divisions (1, 2) of the lateral cutaneous offsets of the first and second intercostal nerves, which emerged from the first and second intercostal spaces respectively.

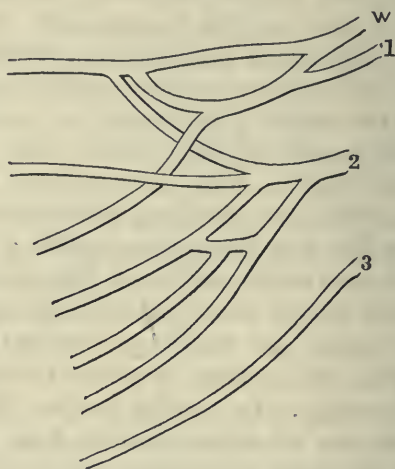
There were no less than seven large nerves passing to the humeral region. The first of these was made up of three branches, one from the nerve of Wrisberg, another from the trunk formed by the first intercosto-humeral and the fasciculus from the nerve of Wrisberg, and a third from the second intercosto-humeral.

The second was a branch of the second intercosto-humeral.

The third was the remaining division of the trunk formed by the first intercosto-humeral and the branch from the nerve of Wrisberg.

The fourth, fifth, and sixth were offsets from the second intercosto-humeral.

FIG. 11.



The seventh was a third intercosto-humeral, which did not join the plexus.

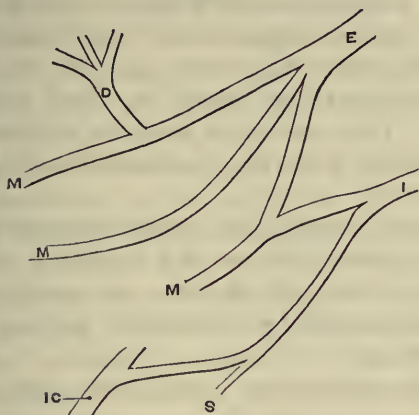
The left side had been damaged before attention was called to it, but it was possible to trace seven similar nerves to the humeral region, though they were made up rather differently. The nerve of Wrisberg made the first and was probably joined by a branch from the first intercosto-humeral, though this had been cut. The second and third were branches from the first intercosto-humeral. The fourth and fifth were from the second intercosto-humeral, the sixth from the first and second intercosto-humeral, and the seventh from the third intercosto-humeral.

The communications between the intercosto-humeral nerves and the nerve of Wrisberg present great variety and are of physiological interest. The plexus just described has been recorded because it was unusually large.

An external anterior thoracic supplying the deltoid is shown in Fig. 12, this occurred in the same subject as the one with the large intercosto-humeral plexuses.

The external anterior thoracic (E) gave large muscular branches (M) to the pectoralis major, one of which (the highest) gave off a large branch splitting into several to the deltoid.

FIG. 12.



The internal anterior thoracic supplied both pectoralis major and minor, and gave a branch to the skin of the arm, which communicated with the internal cutaneous nerve. Similar abnormalities have been recorded in the eighteenth volume of the 'Guy's Hospital Reports.'

VISCERA.

Once the œsophagus was noticed to enter the thorax half an inch to the right of the middle line, but lower down it bent in towards the aorta, its relation to which was normal.

In a subject in which the rectum was on the right side the following was the arrangement:—The ascending and transverse colon were normal, but the descending colon passed diagonally over the front of the fourth lumbar vertebra. It had a short but complete mesentery which bound it down deeply in the abdomen and was attached to the fourth lumbar vertebra and right side of the fifth just below the mesentery of the small intestine. There was no sigmoid flexure, and the mesentery of the first part of the rectum was attached to the sacrum three quarters of an inch to the right of the middle line. At its second part the rectum inclined to the middle line and having reached it, took a normal course to its termination.

This rare abnormality has not been previously recorded in the 'Guy's Hospital Reports.'

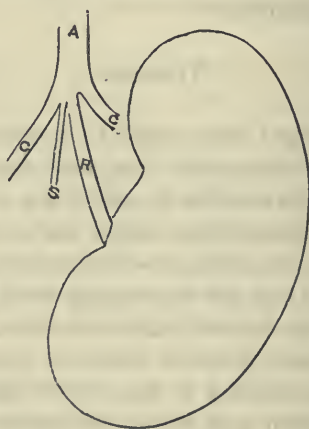
In another subject the right kidney had two distinct ureters, one from the extreme upper end of the hilum and the other from the lower end. They were equal in size but each was rather less than the ureter usually is. Both were behind the renal artery. They converged over the middle of the psoas muscle from which point they continued as a single ureter to the bladder.

A diverticulum from the gall-bladder occurred in one subject. It was rounded and of the size of a hazel nut, coming off from the distal end of the gall bladder. Its opening into the gall bladder was surrounded by a semilunar valvular fold. There were no gall stones.

We have also seen several instances of constrictions of the gall-bladder.

The left kidney was abnormally placed in one subject. Its size was normal. It was placed on the left sacro-iliac

FIG. 13.



symphysis, lying obliquely from above downwards and inwards, with the upper part on the quadratus lumborum, and the lower part projecting into the pelvis. (Fig. 13).

The left renal artery (R) came off from the bifurcation of the aorta (A) close to the arteria sacra media (s) between the two common iliac arteries (cc).

A small artery also entered the kidney at its lower part, from

the left internal iliac artery before its division. A vein and nerves and fascia entered with this small artery.

The ureter was in front. The vein was in the middle but curved round so as to get behind the artery. The sigmoid flexure surrounded the kidney.

Quain states that the kidneys may, one or both, be situated lower down than usual, even in the pelvis. Horse-shoe shaped kidneys have been recorded in our Reports but no instances of alteration in position. The relative position of the ureter, nerve and artery, suggested that the kidney had turned over so that its lower end got highest and its posterior surface anterior.

It is rare for the kidney to receive its artery from the bifurcation of the aorta, and only one case has been recorded where the renal artery is derived from the internal iliac. In our case however, the branch from the internal iliac was only small.

TWO CASES
OF
PULSATILE TUMOUR AT THE ROOT OF
THE NECK.

BY C. H. GOLDING-BIRD,

AND

F. A. MAHOMED, M.D.

FROM THE REPORTS OF MESSRS. W. A. LANE, AND ROBERT H. PERKS.

THESE two cases are offered as a contribution to the literature of pulsatile tumours at the root of the neck—which are not always aneurismal.

Of these two, one was aneurism but was accompanied with peculiar symptoms; the other was a post-sternal abscess which simulated aneurism.

Eugene S—, æt. 38, was first admitted under Dr. Hilton Fagge into Guy's Hospital on May 13th, 1880.

The report of that date is as follows: By occupation a deal porter, and during the last twenty-four years has had to carry very heavy weights. Family history good. Up to fifteen years ago he was perfectly healthy. Then he had typhus and was ill seven weeks. No history of syphilis but he has drunk freely. About eighteen months ago he had a bad attack of bronchitis and he could not work for two months. He

coughed most violently : he had a second attack three months ago, which continues at present.

In January, 1880, he first observed a small pulsating swelling in the supra-sternal notch, and it used to increase in size on coughing, but not nearly so much as at present : and whilst coughing the pulsation in it diminished.

The tumour subsided when the fit of coughing was over. It in no way interfered with deglutition and respiration. Latterly he had been losing flesh.

On admission, there is to be seen in the supra-sternal notch, which appears preternaturally deep, a small rounded swelling pulsatile and expansile. On coughing, which is a very violent act with him always, the lump increases enormously in size, separating widely the two sterno-mastoids and rising upwards over the trachea and cricoid cartilage. When the fit of coughing is over he feels as though a heavy weight dropped behind the right clavicle, and the lump returns to its natural size ; while expanded, pulsation in it ceases.

Percussion over the tumour gives a subresonant note, and at times, when breathing, auscultation indicates pressure upon the trachea, as though the air-passages were narrowed. There is no bruit audible. When dilated its greatest diameter is the horizontal one and it reaches more to the right than to the left.

Along the upper two thirds of the sternum a prolonged blowing sound is heard, sometimes accompanied by occasional rhonchus.

Complaints of pain chiefly in upper part of the right chest and just below the right clavicle. The upper part of chest expands very badly and the right side worse than the left ; the left also is the more resonant. Precordial dulness diminished, and apex beat cannot be felt : the radials beat synchronously.

During his stay in hospital there was no amelioration of his symptoms and he had pain, unrelieved by iodide of potassium and salicylic acid, down the right arm.

He was discharged on June 15th, 1880, but readmitted after a stay at the seaside on July 20th. He then seemed worse : the lump was larger and very painful ; and more so if he stood upright, he therefore walked rather bowed. The pulsation had altered in character. There was not the same increase in bulk as before, but when distended there was great venous conges-

tion of the head and neck and suffusion of the countenance, otherwise the symptoms before detailed remained. An attempt to use the laryngoscope failed; but just before his discharge on Sept. 1st, 1880, a view of the cords was obtained; they were normal, but the cough was now noted to be aneurismal.

Two months later he was again admitted into the hospital, this time under Dr. Wilks. On the whole the patient's condition was much as before, but this note was made on Nov. 18th. Lies without orthopnoea. There is a small rounded pulsatile swelling to the right of the median line, within and behind the right sterno-mastoid muscle. There is no undue pulsation in the right posterior triangle. The clavicle is not displaced. No bruit, but loud tracheal rhonchi. The swelling is dull on percussion and does not move with deglutition. There is no albumen in the urine.

He was discharged on June 14th, 1881, and readmitted for the last time under Dr. Hilton Fagge on Feb. 22nd, 1881. During his absence from the hospital the pain and cough had both much increased, but the size of the tumour is the same as it was at the last report.

A laryngoscopic view showed the glottis normal but the front and right wall of the trachea, about the 5th—6th rings, pushed inwards. The bifurcation of the trachea was visible beyond this narrowed part.

Sphygmographic tracings of the radials were taken (see p. 95, *et seq.*).

On May 29th the patient was seen by Mr. Golding-Bird with a view to an exploratory puncture, but this he objected to do, though he considered it a case in which an exploratory incision down to the tumour might be made with advantage. Other means of relief having failed hitherto it was agreed that an incision should be made, and for that purpose the man was transferred into Lazarus Ward under Mr. Golding-Bird on April 5th, 1881, when the following note was made:

Subjective symptoms.—Pain, worse at times, of neuralgic and spasmodic character, affecting right side of neck and whole of right shoulder, not confined to any one spot, but generally distributed; pain also down the right arm, especially on back and inner side, also on back of forearm and ball of thumb.

Spot of great pain on deep pressure upon clavicular attach-

ment of right sterno-mastoid muscle, and on pressure over sternal end of right clavicle. At times suffocative sensations and a difficulty in "fetching his breath."

Objective symptoms.—Right pulse very small and weak as compared with left; otherwise right upper extremity is normal. All movements perfect and they do not cause pain.

The right subclavian artery in its third part, beats less strongly than the third part of the left, and is a trifle higher in position than the left.

Right brachial weaker than left and rather thready. Right carotid beats less strongly than the left and is less full. Right temporal artery is weaker than the left.

There is no difference on pinching the ears, the right one taking no longer to resume its natural colour than the left.

Seen in a favorable light, the following may be made out. Some bulging forwards of the right sterno-mastoid muscle with loss of depression between its clavicular and sternal origins.

Slight projection forwards of the inner half of the right clavicle but not displaced from sternum. In episternal notch, close to origin of right sterno-mastoid muscle, the skin may be seen to move as though from some pulsating body beneath; this is very faint and only shows in an oblique light, unless he has been straining, when the pulsations are easily seen. There is no visible swelling.

Respiration, when tranquil, is normal, and there is no suffusion of countenance, but forced inspiration is accompanied with a loud metallic tracheal rhonchus. This varies considerably; at times (he states) this comes on when breathing quietly and produces suffocative symptoms. The laryngoscope shows the same bulging inwards of the anterior and right tracheal walls as on a former occasion. Percussion note non-resonant over the inner third of the right clavicle and the same extent of the first intercostal space. No bruit, heart sounds normal.

The finger on the episternal notch, pressed rather deeply against the origin of the right sterno-mastoid muscle, feels the convex limit of a globular pulsatile swelling, apparently of the size of the end of a hen's egg. The beat is synchronous with that of the carotid artery; and the pulsation is not only in the episternal notch, but may be felt also between the two heads of origin of the right sterno-mastoid, and on the outer side of

this muscle. Pressure on the pulsatile spot outside the muscle gives rise to sharp pain. The lateral pulsation is everywhere less marked than the antero-posterior.

On coughing or straining the shape of the whole neck alters remarkably. A globular swelling appears in the episternal notch, apparently rising from behind the right sterno-clavicular joint; the whole of the right side of the neck becomes puffed out, but never to such an extent as to obliterate the prominence of the clavicle. The superficial veins of the right side become very turgid; the left side is in both these particulars but slightly affected. If the breath is held these conditions remain, but if not held, then the neck *gradually* returns to its normal state in about ten seconds. Whilst the turgescence lasts, the episternal (pulsatile) swelling ceases to pulsate; it is dull on percussion.

If told to make a strong expiratory effort with the mouth closed, and to hold the breath for about thirty seconds, the following points are observable. The swelling in the episternal notch rises as before but the general puffiness of both sides of the neck is now so great that it completely conceals the tumour, and so nothing but a uniform puffiness from side to side is to be seen, giving at a short distance the idea of a large goitre, which was limited below by the clavicular line, and which extended laterally under the sterno-mastoids. Accompanying all these appearances, there is great suffusion and venous congestion of the face and both sides of the neck.

On taking breath again the puffiness and swelling subside, that on the left side disappearing first, and last of all the episternal tumour.

A profile view, whilst the breath is held, shows a great convexity of the ordinary profile outline of the throat.

The tumour can be felt to rise slightly in deglutition.

On April 14th, under chloroform, an exploratory incision was made over and above the tumour, in the line of the inner edge of the right sterno-mastoid, as though for ligature of the common carotid. The right sterno-hyoid and sterno-thyroid muscles being divided, a rounded tumour was exposed lying where the right thyroid lobe would be expected to be found; to the inner side of the common carotid artery it was hard, elastic, pulsatile and distensible, evidently aneurismal. Fortunately, the anæ-

thetic produced much the same congestion or coughing, so that the tumour rose one and a half inches above the sternum. The anterior jugular vein was exposed during the operation and divided between ligatures. By its deep surface the tumour was adherent to the anterior and lateral walls of the trachea; but the tissues around were normal and showed no signs of having been inflamed.

In exposing the tumour its overlying structures had been stripped off, that the tumour now seemed very thin walled; it was not even now, however, clear whether it came from the innominate artery or from the aorta.

Mr. Bryant, who was present, thought the latter very likely; and it was at his suggestion that the subsequent step of tying the common carotid was undertaken, without coincident ligation of the subclavian, as he was of opinion that the thin-walled sac would perhaps not resist the increased blood pressure put upon it, if both the arteries were tied.

A ligature was now placed round the common carotid on the right side in the usual way, a slight enlargement of the fresh incision upwards being necessary. The spot chosen was just above the point of crossing of the middle thyroid vein. The operation was not performed under the spray, but the wound from time to time was freely swabbed out with carbolic lotion. Antiseptic dressings were employed. The ligature round the carotid artery was of carbolised gut.

Immediately after the operation the pulse on the right side was fuller and better than that on the left: (a tracing was taken the next day, which will be found described at the end of this case).

The subsequent history may thus be summed up. The wound healed by primary union, except at the lowest part, where a little pus was seen on the dressings on the 18th. After the first twenty four hours he began to complain of tightness of the chest, which loosening the bandages did not relieve, followed by dyspnœa and expectoration of a tenacious mucus. The dyspnœa rapidly increased and he became semicyanotic; the use of the tent, with steam, for a time gave relief. The symptoms however, rapidly augmented; he had orthopnœa and died rather suddenly on the evening of the 20th, from apparently embarrassed breathing.

The difficulty lay evidently in the lung; the larynx and trachea were free.

The temperature chart showed 101.5° as the highest point reached; there was no marked augmentation of the evening over the morning temperature in any one day.

Post-mortem.—Arteries generally atheromatous. Four arteries rose from the arch of the aorta, the abnormal one being the left vertebral, which lay between and rather behind the origins of the left carotid and subclavian arteries.

The innominate artery was rather dilated and three inches long. Arch of aorta very dilated, atheromatous and pouchy, one pouch admitting the top of the thumb. Right subclavian dilated and very atheromatous; right carotid seemed of unusually large diameter but its walls were healthy.

In the left wall of the innominate artery, about its middle, was a sharply-defined oval opening leading into an aneurismal sac about the size of two hen's eggs, which was adherent to, and caused to bulge inwards, the left and anterior walls of the trachea. The opening of the aneurism was vertical, and did not involve either the carotid or the aorta. On cutting into it it was found lined, to the depth of $\frac{3}{8}$ in., with an old laminated clot, and its cavity was partly filled with a soft recent clot, which might have occurred after the operation. The part of the sac yet empty held about an ounce of fluid.

The common carotid, where ligatured, had a soft clot extending to the bifurcation; on the proximal side of the ligature was also a small clot. The carbolised gut ligature still held but was beginning to soften; there was no sign of suppuration. On the parts being removed from the body an abscess was torn open lying next the spine, and between it and the pharynx and larynx. Its presence had never been suspected. The tissues hitherto examined had shown nothing indicative of suppuration. The precise cause of the abscess could not be determined.

In the thoracic aorta was a small aneurism eroding the bodies of the vertebræ.

The brain was normal; the meninges showed slight opacity. The vertebral arteries at the base of the brain were of equal size, and the vessels generally in the base were symmetrical in all respects.

The general history of this case from first to last is undoubt-

edly a fairly ordinary one of aneurism either of the innominate artery or aortic arch; yet when studied in detail there are many points that make the diagnosis uncertain, and indeed prior to the operation different opinions were expressed as to the patient's condition.

Looking at his personal history, his hard life—both in point of laboriousness and of abuse of alcohol—favours the view of aneurism, there being but one unusual symptom during the first six months of his being under observation, viz. up to June, 1880. This was the peculiar way in which the tumour rose from behind the sternum on straining or on coughing, and immediately afterwards subsiding. At a later period it was found to rise on deglutition, and this was afterwards known to be due to its attachment to the trachea, while the former symptom remained unexplained even by the post-mortem examination. Had the aneurism been attached by anything in the way of a pedicle to the artery, it would have been comparatively easy to have explained both the rising on straining and the immediate stoppage of pulsation; but the autopsy showed a long vertical slit opening at once into the artery, there being no specially facile movement of the tumour upon the blood-vessel. The position of the junction of the innominate veins between the chest wall and the tumour quite accounts for the suffusion of the face and neck and evident obstruction to venous return.

In a careful examination made prior to the operation it was observed that the left side was far less congested than the right; but from what was afterwards known of the position of the tumour, the reverse might have been expected; for the tumour lay directed against the left innominate vein.

That the peripheral arterial tract on the right side should be weaker than on the left is only what would be expected in aneurism, but it is worth notice that the ear-test failed in this case. It is the one recommended by Mr. Richard Barwell, and by him personally applied in this case; but the delayed filling of the capillaries after compression of the organ could not be shown to exist. He also in the face of this and the non-pulsatile character of the tumour when "raised," felt a difficulty in agreeing with the diagnosis of aneurism.

Both before and after the operation tracings were taken of the pulse, but these will receive special notice.

The operation presented no difficulty nor anything worthy of special note beyond what has been already detailed.

Nothing in the after state of the patient for a moment suggested the presence of a post-pharyngeal abscess; nor indeed of suppuration at all, and he was believed to be dying solely through pneumonia.

No direct communication existed between the suppurating cavity and the wound, since the latter was fully examined at the autopsy; and it was not until the parts were stripped from the spine that the abscess was found.

James K—, æt. 24, was admitted into Guy's Hospital under Dr. Hilton Fagge, on Nov. 3rd, 1881.

He was by occupation a groom, a thoroughly healthy young fellow, and presenting in all points a clean bill of health. There was no history of syphilis nor of abuse of alcohol.

Present history.—A month ago while shaving he noticed a swelling in the root of the neck, just above the manubrium sterni, and between the two sterno-mastoids; it has not apparently increased in size. In a day or two after first observing it, he had pain in the throat and dysphagia, the chin became puffy and tender and the left side of the neck inflamed. The following day his throat felt "dried up;" he could not swallow at all, but his breathing was normal. He became feverish and his doctor, who then examined him, pronounced his case one of diphtheria, but as an abscess shortly broke in his mouth it is probable he was suffering from quinsy.

He rapidly recovered, and the swelling at the root of the neck remained as before.

On examination, there is over the trachea and just above the sternum a soft, globular, pulsatile swelling, measuring 2×2.75 inches; it lies rather more over to the right side than the left. It rises during deglutition; its upper end reaches the cricoid cartilage. On palpation it is felt pulsating powerfully, but there is no bruit nor thrill; and the lateral expansion is less marked than the antero-posterior. Rather larger at its upper end, the tumour is elastic and feels like a cyst with thin walls, the upper part of which is softer than the lower, but pulsates less markedly. When the head is thrown back the sternohyoid and sternothyroid muscles, like bands, can be felt over the swelling, and pulsation

is completely arrested ; it returns, however, on the chin being lowered. Coughing produces no change in the tumour.

The circulatory system is normal ; and below the tumour there is resonance before the cardiac dulness is reached.

The dulness due to the tumour extends over the upper part of the sternum as far as the level of the cartilage of the second rib, and covers also the cartilage of the two first ribs on the right side ; on the left side the note is quite resonant over the same costal area.

The radial and temporal pulses are both normal on their respective sides. (See sphygmographic tracings.)

On Nov. 8th the patient complained of pain in the swelling, and then also stated that two months ago he had the skin rubbed over the front of the neck by a shirt-stud in the site of the present swelling. There is no mark now on the skin.

On Nov. 11th he was seen by Mr. Golding-Bird, who reported as follows : " In my opinion this is a case of aneurism adherent to the wall of the trachea. It is I believe a lateral tumour and not a fusiform expansion. It is either on some small thyroid artery, or more probably is a saccular aneurism in connection with the innominate artery."

Dr. Hilton Fagge had already expressed an opinion in favour of aneurism, and on 16th Nov. put the patient upon iodide of potassium and low diet, ordering him to keep quite still in bed.

The following day, not liking the treatment, he went out for the day to see his friends but returned within twenty-four hours.

Dr. Goodhart examined him and " thinks there is no inherent pulsation, and that it is not an aneurism ; he thinks there may be a hard hypertrophied piece of the thyroid through which pulsation is conveyed to a cyst connected with the thyroid in front of it."

At this time he was seen by Dr. Felix Semon at the invitation of Mr. Golding-Bird. Although he did not absolutely concur with the view that it was aneurism, yet he was certain as to there being inherent pulsation, and thought that some very vascular tumour, *e.g.* a pulsatile sarcoma, might explain all the symptoms.

Up to this time the skin over the tumour had been normal, and though there had been a slight increase in the size of the tumour, yet pulsation in it had not varied.

On Nov. 19th the temperature rose to 99.2° and the tumour began to be painful. Mr. Davies Colley saw him and gave the opinion that the tumour was not connected with the interior of the aorta.

Nov. 22nd, the skin over the tumour is injected and not so sensitive as that of the surrounding parts.

From this date to Dec. 2nd the patient went home; on his return Dr. Mahomed, the medical registrar, made the following note:

“Dec. 2.—He has returned to the Hospital; during his absence the tumour has increased very greatly and rapidly in size. It now measures $3\frac{1}{4} \times 3\frac{1}{4}$ in., and reaches as high as the lower margin of the thyroid cartilage. It is much more red over the surface, which is covered with dilated small vessels; there is no bruit over the tumour nor in the aorta. The lower part of the tumour is harder than the upper. He cannot now put it on the stretch sufficiently to raise it up when he throws his head back. The skin is shining and apparently very thin, and seems on the verge of rupture. When the pricking and throbbing in it are severe, he relieves the pain by holding a hot sponge to his neck.”

On the same day he was seen by Mr. Bryant who pronounced the tumour to be an abscess.

On Dec. 8th, the swelling and redness having increased, he was again seen by Mr. Golding-Bird in consultation with Dr. Fagge; and it being evidently now an acute abscess, it was at once decided to operate under an anæsthetic. A grooved needle was first thrust in and pus having been drawn, a free vertical incision was made.

An examination of the cavity was now made by the finger; it was found to be bounded anteriorly by the sternum and rib cartilages of the right side, behind by the trachea and its bifurcation, and below the finger rested upon the arch of the aorta, which was distinctly felt pulsating.

The matter that came away—about 3 oz. in all—was partly curdy, partly fluid. The cavity was filled with carbolic lotion, and the fluid could be seen pulsating synchronously with the heart's beat. The drainage tube that was inserted, also moved like the rod of the sphygmograph.

The after treatment of the case was by drainage, and on

general surgical principles. The patient was discharged on Dec. 31st.

At the end of January, 1882, the following note was made at Mr. Golding-Bird's out patients': "Merely a superficial wound now; much episternal pulsation still, but otherwise quite well."

Apart from what the sphygmograph in Dr. Mahomed's hands indicated, there were undoubtedly two circumstances that militated from the first against aneurism; one was the fact that while pulsation was well enough marked in the tumour yet the tension in the sac was clearly not nearly equal to that in the arterial-system; the other, that the peripheral circulation on both sides was, to ordinary observation, in all points symmetrical and normal.

I have no hesitation in saying that, as far as I am personally concerned, I was biassed by my experience of the other case of pulsatile tumour, recorded with this one, and which had come under my observation some months before.

With the exception of the two circumstances just detailed, there was nothing in this case that was absolutely against aneurism; the movement on deglutition, and especially the cessation of pulsation on throwing the head back, having been seen in the other case that proved to be aneurism. In this case of abscess it is not hard to explain the cessation of pulsation. The projection forwards of the vertebral column, on throwing the head back, forced the trachea against the upper edge of the sternum so as to cut off the intrathoracic from the extrathoracic portion of the swelling; pulsation in the latter necessarily therefore ceased for the time.

It is most likely—as Dr. Mahomed thought for some days prior to the tumour being opened—that the abscess was of glandular origin and dependent upon the irritation of the collar stud, of which, however, nothing was known until he had been some time in the hospital.

Note on the Sphygmographic tracings obtained in the preceding cases. By F. A. MAHOMED, M.D.

The assistance which the sphygmograph offered in these two cases is best seen by comparing the tracings obtained in the one with those obtained in the other. Figs. 1, 2, 3, and 4, obtained from the case of aneurism, are typically tracings from a case of aortic disease. The systolic expansion is prolonged, the collapse is sudden instead of sustained, the diastolic expansion is imperfect and insufficient. These tracings emphatically condemn the patient's circulatory system. On the other hand, the tracings obtained from the other case (Figs. 5, 6, 7, 8 and 9) are all typically those of healthy vessels and healthy heart, in fact, those of a normal circulation. No fault can be found with them, though they exhibit some interesting physiological features, which will be described hereafter. This evidence from the sphygmograph is exactly coincident with the general appearance of the patients, and the two largely determined me in forming a correct diagnosis in each case. This I arrived at on the first examination of each, and maintained throughout. The one was a typically "atheromatous" subject, with ill-nourished tissues, thin dry hair, harsh skin, with sebaceous nodules, and that appearance which Laycock has so well described as characteristic of atheroma, while the other was a soft, delicate skinned, ruddy complexioned, healthy-looking lad, not at all the subject for aneurism. Moreover, the low tension of the sac attracted my attention very early in the latter case; the tension of the sac was far less than the normal tension of the arterial system, and therefore of that of all aneurismal tumours.

But to return to the tracings, those from the first case were not taken by myself, and I am not therefore able to vouch for their accuracy in minor details. The general form of them is evidently correct, and it is extremely difficult to get strictly comparable tracings from the opposite sides in cases of this nature. Very little alteration in position or pressure will alter the height of the upstroke. The tale which they all unite in telling is that of diseased vessels. They also seem to show a variation in the size and quality of the pulse on the

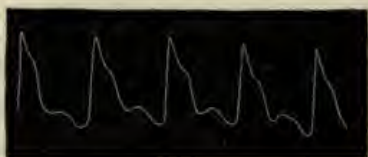


FIG. 1.—Right radial. Pr. 3 oz.
Feb. 28th, 1881. (Before ligation.)

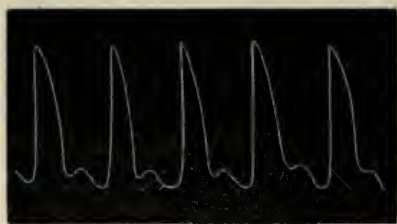


FIG. 2.—Left radial. Pr. 5 oz.
Feb. 28th, 1881. (Before ligation.)

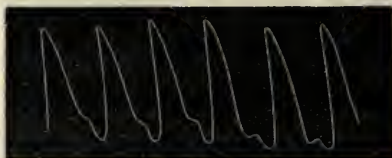


FIG. 3.—Right radial. Pr. $1\frac{1}{2}$ oz.
April 15th, 1881. (After ligation.)

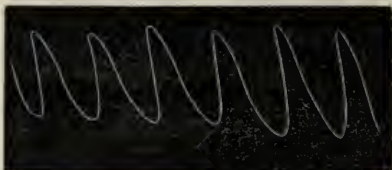


FIG. 4.—Left radial. Pr. $1\frac{1}{2}$ oz.
April 15th, 1881. (After ligation.)

two sides. Before ligation the left pulse seems larger and stronger than the right, for it gives a higher upstroke under 2 oz. more pressure. After ligation the circulation on the two sides seems to have undergone a considerable change, due either to the coagulation which had been induced in the aneurismal sac of the innominate or to increase in the disease of the aorta since the first observation six weeks previously. The right pulse presents no further evidence of aneurism, but the left is now typically aneurismal. It has lost all trace of its secondary waves; the dicrotic, which is well marked on the opposite side, is not visible here, and it approximates to the simple uninterrupted up-and-down stroke which is seen when the aneurismal characters are best developed in a pulse. The tendency of the upstroke to slope backwards is due to the

tracing having been taken on the upper part of a broad slip of paper, the lever of the sphygmograph being raised above the level of its axis, and tending, therefore, to sweep backwards. The chord of the arc which the upstroke forms would no longer be at right angles to a line drawn horizontally through the axis of the lever, as it would be when the lever is working at a lower level. The tracings, therefore, in this case indicated, first, severe aortic disease; secondly, an inequality of the pulses, pointing to aneurism.

The tracings from the other case are those of a normal pulse, with practically no difference between the form or volume of the wave in the two radial arteries. They therefore afford no indication of aneurism; in fact, they do more, they contra-indicate it. The chief proof, however, which the sphygmograph afforded that this case was not one of aneurism, was given by the striking alteration produced in the pulse wave by

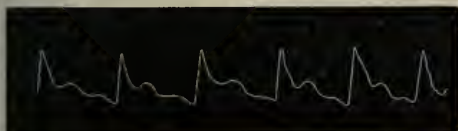


FIG. 5.—Right radial.
Pr. 3 oz. Head down.

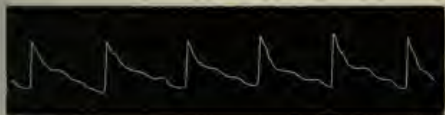


FIG. 6.—Left radial.
Pr. 3 oz. Head down.

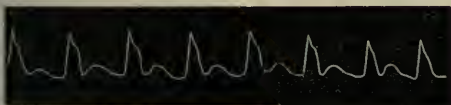


FIG. 7.—Left radial.
Pr. 3 oz. Head thrown back.

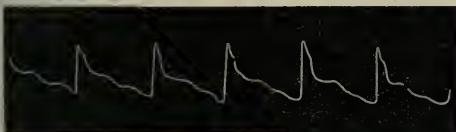


FIG. 8.—Left radial.
Pr. 3 oz. Head down.
Taken immediately after
Fig. 7.

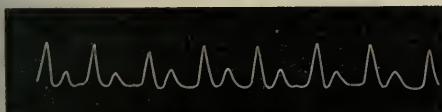


FIG. 9.—Right radial.
Pr. 3 oz. Head thrown back.
Taken immediately
after Fig. 8.

the change in the position of the head. When the head was held in the ordinary position the tracings reproduced in Figs. 5 and 6 were obtained. The head was now thrown back as far as possible with the view of raising the tumour out of the thorax ; in fact, of lifting it off the aorta, and the tracing seen in Fig. 7 was produced. The pulse immediately became very dicrotic. The head was now restored to the normal position, and the pulse returned to its previous form (Fig. 8). The experiment was repeated, the tracings being next obtained from the right side, with exactly similar results. Fig. 9 represents the pulse on this side with the head thrown back, and the next tracing obtained was a recurrence to the normal with the head in the ordinary position. These observations were repeated several times with the same results. I also made corresponding observations on healthy persons, but without producing any marked variation in the form of the pulse wave by changes in the position of the head. The explanation which suggested itself to me of these changes was that the sac of the abscess rested upon the transverse part of the arch of the aorta, and acted as a sort of "damper" to its movements. When the head was thrown back the sac, I take it, was somewhat raised from the aorta, and allowed the dicrotic wave, which originates in the aorta itself, to pass towards the periphery ; when the head was depressed it pressed down upon the aorta and arrested this wave. If this explanation be true it would seem to prove that the dicrotic wave is a centrifugal one. On the other hand, an objection may be offered to this explanation on the ground that not only is the pulse made dicrotic by the altered position of the head, but it also becomes more rapid ; and it may be argued that the mere obliteration of the dicrotic wave would not of itself increase the rapidity of the pulse, that probably some other effect is produced on the cardiac ganglia, which accounts for both phenomena in a less mechanical way. To this criticism I would reply that it has been shown experimentally, and observed clinically, that diminution of arterial pressure increases the frequency of the heart beats, while increased arterial pressure diminishes their frequency. Of course this result is produced through the cardiac ganglia by the diminution and increase of the effort required to open the aortic valves.

Now, it appears probable that the pressure of this abscess cavity upon the top of the aorta would considerably impede its expansion, and therefore to some extent increase the effort required of the ventricle to enable it to empty its cavity in the required time. Throwing the head backwards by raising the sac from the aorta, and removing this impediment, would therefore produce the same effect as diminishing the general arterial pressure. It would, therefore, increase the frequency of the heart beat.

Though I venture to offer this explanation of the facts observed I cannot regard it as proven. Whatever may be the correct interpretation of the facts, they would seem conclusively to indicate that the tumour was external to the aorta, and could be made to alter its relation to the vessel by changing the position of the head.

I may add that I ascertained, whilst this case was under observation, by the examination of several bodies, that a lymphatic gland rests upon the arch of the aorta, and lies between the innominate artery and left carotid. This gland I regarded as the probable seat of the abscess, which I always believed to exist.

THE SURGICAL AFFECTIONS OF THE TONGUE.

By THOMAS BRYANT.

A REFERENCE to the past numbers of these 'Reports' shows that diseases of the tongue have received but scanty notice from my predecessors; and I am disposed to think that an explanation of this fact is to be found, rather in the difficulty that is experienced in making the subject intelligible without coloured drawings, than in any want of interest in the diseases of an organ which holds such an important position as the tongue in the animal economy.

With this feeling, I purpose, in the present paper, to do something towards filling the gap, and to illustrate by cases where I can, and by drawings where they are available, the different diseases of the tongue as seen in the practice of the general surgeon.

I shall, in doing this, record examples of *congenital* affections of the tongue, including cases of hypertrophy and of *nævi*, as well as of tumours. Also cases of inflammation, ulceration, and suppuration arising from idiopathic and traumatic causes, as well as from the presence of a hydatid cyst.

I shall discuss the syphilitic diseases of the tongue, as well

as the cancerous, and show how the former disease, when persistent or relapsing, leads up to the latter.

The disease known as ichthyosis of the tongue will receive special notice as well as special illustration, and the close connection between it and cancer will be dwelt upon.

The subject of tubercular disease of the tongue will then be illustrated, and other cases of tumours given, including examples of cystic tumours of the tongue, adenoma, a doubtful case of true cancer, and of aneurism by anastomosis.

CONGENITAL AFFECTIONS.

Hypertrophy or macro-glossia is without doubt a congenital affection, although in certain reported cases it may not have been observed till the first or second year of life. It is usually an affection of slow growth, and troublesome on account of the mechanical obstruction it causes to deglutition and speech: when the disease has existed for years it produces deformity of the teeth and jaws from the local pressure of the tongue upon the former and the nonclosure of the mouth. Syme published a case in which the tongue projected out of the mouth of a girl, aged 14, for three inches; and Humphry another, in a child, aged 11, in which when the tongue was withdrawn into the mouth as far as possible, the exposed part measured, from the upper lip to its tip, two inches. Many other cases are also on record. As a rule this affection involves both sides of the tongue, but in exceptional cases it may affect but one. The growth is generally painless and the disease not rarely affects idiots and children with ill-formed crania.

CASE 1.—In this case, from which fig. A, Plate I, was taken, the disease was congenital and confined to the right half of the organ. It occurred in a boy who, when six years of age, was admitted into Guy's Hospital, under the care of the late Dr. Thomas Addison, with his tongue protruding far out of his mouth and obstructing respiration. His mother stated that the tongue had been affected ever since his birth;

and that he had never been able to articulate distinctly. Whenever he took cold the tongue became swollen, blisters formed upon it, which burst and bled. The increase of the disease had been gradual. He was treated with mercurials, and he derived so much benefit from them that he left the hospital with the tongue fairly retracted. He reappeared, however, three years later (1856), with the same disease, the growth having rapidly increased for one month before his admission ; he then came into the hands of the late Mr. John Hilton. At that time the right half of the tongue with the submaxillary glands was much enlarged, the whole organ was protruding from the mouth, and the papillæ on the affected side of the tongue were much hypertrophied. He was again treated with half-drachm doses of the solution of the perchloride of mercury and left relieved. In another three years the tongue had grown as large as ever, and he was readmitted for the third time, when the tongue presented much the same appearance as it had on his previous admission, although the papillæ seemed coarser. He was treated in the same way as on the two previous occasions and he left relieved. No further history of his case is known.

The drawing (fig. A) was taken after his second admission into the hospital.

It should be mentioned that in this case there were no symptoms of the presence of næroid tissue. The tongue appeared coarse, thickened, and enlarged, as if from simple muscular hypertrophy, and yet from the fact that the enlargement diminished under the influence of mercury, there must be a question as to this being its true nature. Indeed, the case appears pathologically to be like one shown at the Pathological Society in 1872 by Mr. H. Arnott, in which, after its removal, there was visible microscopically very little true muscular hypertrophy of the organ ; but the epithelial covering of the tongue was very thick, and the papillæ enlarged ; the blood-vessels were larger than usual, and there were large irregular spaces with thin walls, which were filled with blood or clear fluid ; a few vesicular bodies, which may have been enlarged lymphatics, were also present. Macro-glossia, as a disease is, according to Mr. Arnott, probably due to a variety

of causes, that is, to (1) a true muscular hypertrophy of the organ; (2) a nævoid affection of its blood-vessels; (3) a thickening and induration caused by a long-continued sub-inflammatory state; or (4) a general enlargement of the lymphatics of the tongue.

The treatment of this affection has hitherto been excision, either of a wedge of tongue, as successfully performed by Humphry, or the removal of the projecting portion of the organ by the knife or *écraseur*. But mercurial treatment should certainly be employed in some cases before recourse is had to surgical interference, since in the case I have recorded the benefit of the drug was most striking.

In some cases of enlargement, and more particularly when the disease is not congenital but acquired, and consequently probably inflammatory, the effect of the iodides should also be tried. In the following case it proved valuable.

CASE 2. Hypertrophy of tongue successfully treated with the iodide of potassium.—A gentleman, *æt.* 20, consulted me some years ago for enlargement of the tongue of twelve years' standing, and which I, from its history, regarded as inflammatory. The enlargement was associated with a protrusion of the organ and all the consequent evils. Iodism, induced by ten-grain doses of the iodide of potassium three times a day, was followed within a week by the speedy disappearance of the affection, though at the time, death appeared imminent from the excessive swelling of the organ caused by the drug.

I should like to mention that so early as 1807 Sir A. Cooper removed a portion of a tongue weighing on removal 2 ounces $2\frac{1}{2}$ drachms (Troy), and measuring $3\frac{1}{2}$ inches in length, 3 inches in breadth, and $1\frac{1}{2}$ inches in thickness, from a man *æt.* 53, who had been troubled with the enlargement for six months.¹ The disease was supposed to have been brought about by the use of mercury given for syphilis. The case did well.

ON GROWING AND DEGENERATING NÆVI OF THE TONGUE.

These cases are not very common, and out of about half a

¹ Vide Prep., Guy's Museum, No. 1670.

dozen that I have seen the following (Case 3) is the best. Fig. B, Plate I, was taken from it. I first saw the case when the girl was an infant, and the tongue presented the appearance and feel of a vascular sponge. The whole organ at that time was swollen, and large distended veins coursed over and under its surface, more particularly on its right side; it had likewise a very full arterial supply. On the application of pressure by means of the thumb and fingers the tongue was readily emptied of its blood, and on its removal, it at once refilled. The case was brought to me for treatment, I advised that nothing should be done, and I did so on the recognition of the fact that *nævi* have a tendency to undergo degenerative changes, and in the hope that these changes would take place in the tongue. In this hope I was not disappointed, for during the twelve years that have passed since I first saw the case many changes have taken place in the part, but the most typical is the cystic degeneration of the *nævus*. These changes began when the child was about six years of age, and have steadily continued. At present the tongue has quite lost its spongy feel. In consistence it is tolerably firm, but it feels harder in some spots than in others. To the eye its surface looks to be made up of vesicular warts, these vesicles being filled either with clear or more or less blood-stained serum. In fact the tongue appears precisely as any *nævus* appears which has undergone the peculiar cystic warty degeneration to which such growths are prone. It has, however, probably less of the papillary hypertrophic growths on its dorsum than many *nævi* of mucous surfaces show. These appearances are confined to the upper surface of the tongue, for its lower aspect (Fig. c, Plate I) still presents, in a degree, the venous engorgement which originally characterised the whole growth.

The appearances I have described and illustrated are absolutely typical of a degenerating *nævus*, since no other growths ever undergo like changes nor present any like features.

I must mention, here, a curious *complication* which presented itself in this case, and which is difficult to explain, though I am disposed to think it had something to do with obstruction of the lymphatics. It appeared when the child was ten years of age, and when the cystic degeneration

of the nævus had far advanced ; it showed itself as a painless swelling of the neck, which began in the right submaxillary region and descended backwards towards the angle of the jaw, and downwards along the neck. When I saw it there was a soft, flaccid, baggy enlargement of the part, without any external or general signs of inflammation. I looked upon it as cystic, and advised its removal. On attempting to carry out this practice I found no signs of a cyst wall, but simply a collection of a thin, watery, but highly albuminous fluid in the deep connective tissue of the neck. Indeed, when an incision had been made into the swelling, and the fluid was evacuated, I never saw nor made a more perfect dissection of the submaxillary and digastric spaces than then showed itself. The fluid had clearly been poured out into the connective tissue of these deep spaces, and there was no cyst wall. I washed the cavity out with iodine water and introduced a drainage-tube, and under the kind care of Dr. O'Meara, of Sutton Bridge, Lincolnshire, the case subsequently did perfectly well, and no return of trouble has taken place.

Fig. B, Pl. I, was taken when the child was twelve years of age, two years after the disappearance of the above complication.

It is quite possible that at the present time the tongue of this child, being large and coarse in appearance, might be mistaken for a case of macro-glossia or one of the forms of hypertrophy. No one, however, who recognises the peculiar cystic appearance of its surface should mistake it or fail to recognise its nævoid origin.

Such cases as this have doubtless been described by authors as examples of vesicular disease of the tongue.

Nævi of the tongue do not, however, more than nævi of other parts, always undergo degenerative changes, although when they do, they for the most part assume the appearances presented in the drawing, with such modifications as of necessity result from the surface being cutaneous or mucous. Such nævi will probably require treatment.

CASE 4.—In 1875 I was consulted by Emma C—, æt. 22, who had had what was clearly a nævus on the right side of her tongue from birth. When I saw it, it was about the size

and colour of a black cherry. It could readily be completely emptied. It was troublesome because it was constantly getting between her teeth and being bitten.

On this account, I applied two ligatures to its base, including in each ligature half the nævus with a portion of the tongue, and a good recovery ensued. When nævi of the tongue require treatment the above method is probably the best to adopt.

CONGENITAL TUMOURS OF THE TONGUE OTHER THAN NÆVI.

These do occur, though rarely, and they may appear as outgrowths, warty or otherwise.

CASE 5.—On March 13th, 1864, I was consulted by Alice B—, æt. 14, for a warty growth on the posterior half of her tongue, about the median line, covering the surface to the extent of a sixpence. It had been noticed since her birth, and it had grown slowly. The growth was evidently warty, and sprang from the surface of the tongue. I destroyed the surface with a caustic, and a complete recovery took place in three weeks.

CASE 6.—In January, 1869, Eliza B—, æt. 3 months, was brought to me with a congenital tumour, the size of a large rice seed, beneath the tip of her tongue. It was seen soon after birth and had grown. It was clearly an outgrowth, and had a white appearance. I cut it off, and found it to be fibro-cellular.

I remember, also, some years before seeing this last case, cutting off from the dorsum of an infant's tongue a pedunculated fibro-cellular congenital growth, the size of a pea, and a good recovery followed.

In removing these pedunculated growths it is well to cut well into their bases, since cases have been recorded in which a return after removal has taken place.

Congenital tumours of a deeper kind may likewise occur, though none have come under my notice; a remarkable instance of such was recorded by Mr. Hickman in the

twentieth volume of the 'Pathological Society's Transactions,' in which an infant, sixteen hours after birth, was suffocated by a growth on the base of the tongue, made up of hypertrophied racemose glandular structures normally existing in the part.

Amongst the congenital tumours of the tongue must likewise be mentioned the existence of gummata in the subjects of hereditary syphilis.

INFLAMMATION AND SUPPURATION OF THE TONGUE.

Inflammation of the tongue, when deep seated and general, is a grave affection, since the swelling which accompanies it is often so sudden and severe as to threaten life by suffocation. Such cases are, however, rare; I have seen but one.

I say this, excluding from consideration those cases of sudden enlargement of the tongue the result of salivation from mercury or iodism as in a case already reported (Case 2). In these the symptoms, though severe, as a rule subside rapidly, under local treatment, on the removal of their cause.

In rare cases, however, the tongue may slough off after ptyalism.

Inflammation of the tongue, when local, may be acute or chronic. It may follow an injury, as in Case 7, or come on without any other assignable cause than cold or exposure. It may begin as a sudden swelling of one half of the organ associated with constitutional symptoms of fever, &c., or it may show itself simply as a chronic enlargement of the part with few, if any, general, and no more local symptoms than are to be explained by the mechanical enlargement of the organ.

The disease, however, under both circumstances, is not dangerous; since it is well amenable to treatment, and has a tendency towards recovery. I have seen many examples of this affection and in all a good result took place.

The following are the brief records of such of the cases as I have preserved.

CASE 7. Abscess of tongue following an injury received three months before.—Ellen H—, a healthy-looking child, æt. 2½,

was admitted into Lydia Ward, Guy's Hospital, under my care, on August 23rd, 1875, with a hard, oval-shaped swelling, the size of a large almond, in the right half of her tongue. It had been gradually coming for three months after a wound of the part from a piece of slate pencil.

The swelling had been painless, was hard, and apparently cystic. I punctured it with a lancet, and evacuated a teaspoonful of pus, and a rapid recovery took place.

CASE 8. Acute inflammation of tongue treated by free puncturing; no suppuration; recovery.—Alfred R—, æt. 9, was brought to me at Guy's Hospital on October 3rd, 1864, with an acute enlargement of the right half of his tongue which had come on two days before after exposure to night air. The boy was ill from fever, and low from want of food. The tongue was very œdematous. I punctured it freely with a lancet with marked and rapid relief and gave salines. In one week the symptoms had disappeared and the boy was well.

The benefit of puncturing the acutely inflamed organ was very marked in this case.

CASE 9. Acute abscess in right half of tongue; recovery after opening abscess.—Luke G—, æt. 37, came under my care on September 5th, 1860, with great enlargement of the right half of his tongue. It had been coming on for one week with pain and difficulty in swallowing. He felt as if his tongue were too large for his mouth. The swelling was confined to the right side of the organ, and was fleshy to the feel. There was some general fever. I ordered salines and milk food.

On September 13th, when I saw him again, the swelling was less, but more localised and fluctuating. I introduced a lancet into the organ and let out some pus. After this a rapid convalescence ensued.

CASE 10. Inflammation of the whole of the tongue followed by suppuration; cure.—Henry C—, æt. 37, came under my care on February 3rd, 1862, with a tongue which had been rapidly enlarging for five days, and now filled the mouth. The glands beneath the jaw were likewise enlarged, and

mucus flowed freely from the mouth. It was clearly a case of inflammation of the organ. On the following day an abscess burst and rapid recovery ensued.

CASE 11. *Chronic inflammation of the whole tongue; recovery.*—Rebecca M—, æt. 37, married and three children, came under my care on April 3rd, 1865, with swelling and inflammation of the whole tongue of three months' duration. The tongue was thick, swollen, spongy, glazed, and smooth, and had a very red, raw aspect. There was no history of syphilis. Under the internal and external use of chlorate of potash, with milk and slop diet a good recovery took place.

CASE 12. *Acute inflammation of the right half of the tongue; recovery without suppuration.*—F. E—, æt. 22, came to me on February 7th, 1870, with an acute inflammation of the right half of his tongue of three days' standing. It had come on, without any known cause, with pain and swelling; the part was greatly enlarged and looked tense; there was also some enlargement of the submaxillary gland. The swelling rapidly subsided under the internal use of saline purgatives and locally of acupuncture.

Treatment.—When acute inflammation attacks the tongue as a whole, and threatens life by suffocation, free puncturing or free incisions made in a vertical direction into the organ may be required,—these openings being made with the view of relieving mechanically the turgid conditions of the vessels and of giving exit to the inflammatory fluids which infiltrate the part. Serious hæmorrhage may, however, at times, follow these incisions, and in a case I witnessed of the late Mr. Poland's the result was nearly fatal.

In more local inflammations, the benefit of puncturing the swollen part is very great,—in the early stage to let out the serous fluids, and in the later to let out pus. In the cases given this fact is well illustrated.

By way of medicines salines and tonics are beneficial, but the disease has a tendency to get well by natural processes.

HYDATID CYST IN THE TONGUE GIVING RISE TO SUPPURATION.

The possibility of a chronic cystic enlargement of a tongue, as of other parts, being due to the presence of an hydatid should always be in the mind of the surgeon; and more particularly when the enlargement is painless, and gives rise to trouble mainly from mechanical causes. Also, when a chronic, painless globular tumour has existed in a part for some time, say months, and then suddenly increases—the possibility of the swelling being due to the presence of an hydatid which has died and given rise to suppuration should be entertained—for hydatid tumours in their early stages, in the tongue as elsewhere, give rise to symptoms of a mechanical kind, and at a later period when they die, to suppuration.

I have seen two cases of this affection; one (Case 13) occurred in the person of a middle aged patient, who had a chronic cystic enlargement of one side of the tongue. When the cyst was punctured a globular hydatid escaped and a good recovery ensued.

The second case (Case 14) occurred in 1881, in the person of a girl, æt. 17, who came to me with a *central* cystic swelling of the tongue of seven or eight months existence. The enlargement had been quite painless and felt like a tight globular tumour embedded in the tongue.

I punctured the swelling with a lancet and evacuated a collapsed hydatid cyst floating in pus, and a good recovery took place.

In the first case related the hydatid was turned out entire; in the second, the hydatid had died and had given rise, as any foreign body might, to suppuration. A cure in both cases took place as soon as the foreign body was removed.

CHRONIC SUPERFICIAL GLOSSITIS OR SMOOTH, GLOSSY TONGUE.

A smooth glazed tongue is often met with in practice, and there can be little doubt as to its being the result of a chronic inflammation of the mucous membrane of the organ. At times it is associated with ulceration. This inflammation is in

many cases due to the heat or irritating influence of a hot pipe, cigar, or spirits.

It is well described by Mr. F. Clarke ('Diseases of Tongue,' pp. 159—161). It shows itself in patches more or less oval or oblong, of a deep red colour and raw aspect, the other portions of the tongue presenting their natural appearance. The surface of these patches is smooth and glossy, though at times ulcerated. The tongue itself is occasionally swollen, and where the disease has existed for some time the patches feel thickened and as if elevated. Should the disease be checked in its progress a complete recovery may ensue, but more commonly the patches remain smooth and shining, or become the seat of a white patch.

In preparation 1672⁷⁵, Guy's Museum, there is an interesting example of the affection which occurred in a man, æt. 49, who was admitted with pemphigus and erysipelas, in March, 1878, and who gave a clear history of syphilis five years previously.

The preparation, as described by Dr. Goodhart, shows that the tongue was changed in appearance completely. Its surface, in place of being rough looking, had lost all its papillæ, even the circumvallate, the whole being scarred over with smooth cicatricial tissue. The mucous covering of the tongue was thicker than normal, smooth and white. At two spots were ulcers, one the size of a threepenny piece with an indolent, unhealthy surface, the other larger and more superficial, healing. The tongue was not fissured.

Microscopically these patches "are either entirely denuded of epithelium or it is reduced to an extremely thin layer, and the papillæ are obliterated by distension. Pathologically the disease "appears to be a chronic inflammation of the mucous membrane which has gradually produced complete alteration in the characters of the epidermis and thickening of the corium and submucous tissue." Butlin, "Med. Chir. Trans.," vol. 61.

The disease is constantly the precursor of a cancer.

ULCERATION OF THE TONGUE.

In a clinical point of view it is expedient to divide the

ulcers of the tongue into the superficial and deep—since in a general sense the superficial are local, simple and readily curable; whereas the deep—which are due to the breaking-down of inflammatory, tubercular, syphilitic, or cancerous elements—are complicated, difficult to diagnose and treat, and moreover are dangerous.

The *superficial* sores include the aphthous and dyspeptic ulcers; those associated with chronic glossitis; ulcers excited and kept up by decayed or ragged teeth, as well as some due to syphilis, congenital, or acquired.

The *deep* ulcers are always either syphilitic, cancerous, or tuberculous.

SUPERFICIAL ULCERS.

The ordinary *aphthous* inflammation of the tongue is a common affection, and is met with in children and adults as a result of irritation of the stomach or intestines from dietetic or other causes. In feeble subjects the white aphthous spots may ulcerate and thus become the source of much trouble, and the ulceration may be extensive though rarely deep. In cachectic patients the parts may slough.

The *treatment* of these cases must be mainly determined by the cause; but in the majority, a lotion of chlorate of potass, or boracic acid of five grains to the ounce of water, and the internal administration of the same drugs with or without bark or the mineral acids is generally sufficient to bring about a cure, though in feeble subjects this may be slow.

In the more limited affection, the local application of the nitrate of silver often acts very beneficially; and in the more chronic, quinine is of great value.

THE SIMPLE OR DYSPEPTIC ULCER OF THE TONGUE.

This form of superficial ulcer of the tongue may be the sequel of the aphthous but more commonly it begins as an ulcer, a result of chronic glossitis which steadily spreads. It rarely, if ever, dips into the muscular tissue of the organ,

but is confined to the mucous membrane covering it. The ulcer may be inflamed, indolent, sloughing, or irritable, indeed it may vary as may any ulcer in another part of the body, and if chronic it will be indurated. Its surface, however, will almost always be smooth, and it will never display the irregular, or deeply-excavated appearance of the deep sores—it will, moreover, almost always be seen upon the dorsum of the tongue, although in exceptional instances it may spread downwards; as in the following example.

CASE 15. *Simple or dyspeptic ulcer of the tongue involving both surfaces.*—Mr. L. C., a banker's clerk, æt. 20, came under my care on June 28th, 1860, with extensive superficial ulceration of the tongue of thirteen months' standing; it involved the surface of the organ as well as the soft parts beneath, and had the appearance of aphthous ulceration. It had been gradually spreading during the thirteen months, although at times it had improved and partially healed.

There was *no* glandular enlargement, nor rough teeth. His general health was good, but he said he always suffered from indigestion. He had never had syphilis. I put him on almost milk diet, and prescribed some chlorate of potash internally as well as a lotion. In two weeks the ulceration had much improved and in three it had healed. In six he thought he was well, consequently he took greater liberty in his diet and so had a relapse. He then returned to his careful form of living and in February, 1861, was well.

This patient had been teased overmuch by the much-to-be-condemned, common routine practice, the local application of nitrate of silver and other irritating local applications. He had, moreover, been given mercury in frequent doses under the false impression that the disease was syphilitic. In fact he had suffered from a want of a correct diagnosis of the nature of his case, and had been maltreated as a result.

CASE 16.—In a patient under my care years ago who had habitually passed lithic acid in such abundance as to supply weekly my class at Guy's with specimens, the ulcer had existed for years and was indolent and superficially indurated. It was clearly due to gastric causes. By placing him on milk

diet with alkalies, and using soothing, not irritating, local application, a good recovery took place, although after many months' treatment.

These two cases are the most typical I have seen of the chronic simple dyspeptic ulcer, and they fairly represent the nature, appearance, and treatment of the affection.

Treatment.—The only sound principle of treatment is a soothing one, and the chief good is to be gained by means of diet. This is to be regulated in the most careful manner and all food should be forbidden that can possibly irritate. Milk food, when it can be taken, is the best, and with it, it is well to give alkalies, such as lime water, bicarbonate of potash or chlorate of potash. Animal broths are beneficial—but little meat should be allowed. All beer and spirits should be interdicted, and when stimulants are absolutely necessary they should be given as wine well diluted.

Locally the lotion of boracic acid or chlorate of potash is beneficial, and caustics are rarely required. Of tonics the barks are the best form, but at times the mineral acids are of value.

ULCERS ORIGINATING FROM LOCAL IRRITATION.

These are very common in the tongue, and the fact is due to the restless mobility of the organ, and the necessary friction which it must receive from any sharp process of a broken or decayed tooth or any edge of rough tartar, or even from the presence of an exostosis of the lower jaw, for I have seen such a case which was looked upon as cancerous.

These cases are at times very troublesome and obstinate unless their cause is recognised, and they may, and do, without doubt, often become cancers.

They may show themselves as mere blisters, or superficial ulcers—the other part of the tongue being healthy—but in chronic cases the local sore may be indurated and thus simulate a cancer. In one of the two following cases the ulcer had existed two years.

CASE 17. *Ulcer of tongue of two years' standing.*—Jane W., æt. 40, came under my care at Guy's on Feb. 16th 1865, with an indurated ulcer on the right side of her tongue of *two years' standing*. It was about the size of a sixpence. A sharp tooth was in close contact with it. This was removed and the ulcer rapidly healed.

CASE 18. *Simple Ulceration of Tongue from the irritation of a tooth.*—Eliza C., æt. 53, came under my care at Guy's Hospital on May 13th 1861, with an indurated circular ulcer on the left side of her tongue of one month's standing. It had been regarded as a cancer by another surgeon and excision had been advised—on that account she came to me. The ulcer was circular and somewhat indurated but not raised, it was clearly in close contact with a badly decayed and rough tooth.

The tooth was removed and a lotion of chlorate of potash given, and in one month the tongue was well.

Some subjects are more susceptible to irritation than others, and I have known a patient have repeated blistering and ulceration of the tongue from the irritation of a rough decayed tooth after sleeping on the affected side. The mere weight of the tongue against the tooth and the unconscious friction between the two parts in contact proved enough to produce a blister in the soft parts.

Treatment.—The mere knowledge of the cause of this affection suggests the remedy—namely the removal of the cause—the removal of the point of irritation when possible, and the extraction of the tooth when nothing less is sufficient. Indeed as a general rule of practice the surgeon should always advise the removal of any local source of irritation from the tongue as from any other part of the body—for such is without doubt the cause of the majority of local cancers.

INFILTRATIONS AND DEEP-SEATED ULCERS OF THE TONGUE.

I have of necessity grouped these cases together, for the majority of the deep-seated ulcers of the tongue begin as local

infiltrations and are due to the subsequent breaking down of the infiltrated and infiltrating material.

The group consequently includes cases of syphilitic disease, of epithelial cancer, and of tubercular disease of the tongue, each of which claims a distinct notice.

SYPHILITIC DISEASE OF THE TONGUE.

This shows itself in many ways and under many circumstances. Thus it may occur as a *congenital affection* and appear either as a mucous patch on the tongue associated with other constitutional symptoms, or as a deep fissure as recorded by Dr. Barlow—'Path. Soc. Trans.' vol. 31—or as a superficial ulceration. Of the former kind I have seen several examples; of the latter but one and that through the kindness of my colleague Dr. Goodhart in whose practice it occurred.

The drawing (Plate III, fig. 1) was taken from the preparation now in the Guy's Museum and the following are the notes of the case.

CASE 19. *Congenital syphilis with ulceration of the tongue; laryngitis, pneumonia; death.*—Wm. M., æt. 4 months, was admitted into Lionel Ward, Evelina Hospital, on April 1, 1882, under Dr. Goodhart.

He was one of six children, all the others being healthy. The mother had, however, three miscarriages between the last 3 children. He has always been an ailing child, but was fairly well, up to a month ago when the roof of his mouth became sore, producing a sore nipple in his mother, he was then fed on biscuits and bread, and has wasted ever since. The "Snuffles" were noticed at the same time as the mouth became sore, and a slight punctiform eruption over the nates and scrotum. He has had diarrhoea during the last month and occasionally vomits.

Present state.—Pale and emaciated, skin dry and scurfy, purulent ozæna, decided snuffles and breathing of a croupy nature; no cranio-tabes, fontanelles rather depressed. Rash on nates and scrotum consisting of clean punched out dry sores, one on each side of the anus and one on the right side of scrotum;

also large brown discolorations in various places but chiefly over the scrotum. Weight, 8lb. 10 oz. Thrush over roof of mouth, bleeds when detached; gums pale; throat apparently normal. Heart and lungs are healthy, no enlargement of liver or spleen.

The child was collapsed on admission. Brandy was given. Sores at anus dressed with powdered iodoform.

April 2.—Temp. 101.6° during night, child slept well, breathing rather bad at times, relieved by turpentine stupes. Warm bath at 7 a.m., temp. being 103.6° this produced a fall to 97° .

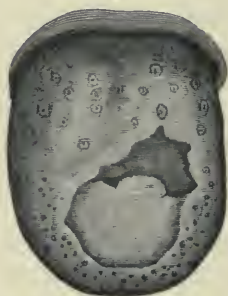
3rd.—Temp. 102° ; pulse 160; skin hot and dry; takes milk $\frac{1}{3}$, with water $\frac{2}{3}$, well; no sickness; breathing easily. Ung. hydrarg. applied on flannel. There is loud bronchial breathing over the left base as high as the angle of the scapula, but no decided dulness. Vapour bath given of calomel gr. viii.

5th.—Vapour bath has been repeated, and hydrarg. c. creta gr. j, at night in addition. Rash on nates much improved in appearance. Breathing became oppressed towards evening and difficulty in swallowing appeared. Slightly convulsed; died quietly at 12.30 p.m.

Post-mortem.—Body emaciated; on comparison of the two knees the left is seen to be distinctly enlarged just above the epiphysis; on removing the soft parts, the periosteum is adherent and coated externally by fatty looking material; on vertical section of the bone, a mass of white new bone was seen surrounding the lower part of the femur immediately above the cartilaginous epiphysis; a section of the other femur was made in the same way and nothing of the kind could be seen. No enlargement of any other bones could be detected.

Tongue.—A semilunar ulcer exists on the posterior third of the dorsum in the median line, with slightly thickened edges. *Larynx*: slight œdema of aryteno-epiglottidean folds; on opening the larynx by a longitudinal section made down the posterior wall, a vertical ulcer was seen about $\frac{1}{4}$ inch long and $\frac{1}{8}$ inch wide, perforating the thyro-hyoid membrane and leading into a cavity of some size in front of it, beneath the muscles, the walls of which were thin and membranous; trachea appeared normal. *Lungs*: considerable consolidation of left lower lobe, due to combined collapse and broncho-pneumonia. On the right side

This figure represents the one referred to in page 117 as
Plate III, fig. 1.



SYPHILITIC ULCERATION OF TONGUE IN INFANT.

the lower lobe was solidified, chiefly from collapse, but with a few patches of broncho-pneumonia. *Liver* large but normal. No reaction with iodine. *Spleen* and *kidneys* normal.

Calvaria healthy, brain substance normal, the membranes over the pons were a little woolly-looking as if indicating early meningitis. Vessels appeared to be perfectly normal.

As an acquired disease, syphilitic disease of the tongue shows itself either as a mucous patch, or as a more or less extensive local infiltration of the tongue with gummous deposit, the breaking down of which leads either to superficial sores or to fissures (*vide* Plate I, fig. E), or to deeply excavated irregular ulcers, and later on to the indurated and irregular cicatricial tongue (*vide* Plate I, fig. D).

In some instances the gummous material is either poured out soft or as a solid which soon breaks down; under both circumstances appearing as a cystic enlargement in the body of the tongue. I have seen, in some cases, four or five of these cystic swellings in a tongue thus affected, and on opening the same have given exit to a thin fluid. The enlargements appear as single or multiple globular tumours in the body of the tongue.

When the disease occurs in the shape of *mucous patches*, it is usually associated with other symptoms; the patches are commonly multiple, and are for the most part situated on the upper surface and edges of the organ. They appear either as moist papules with whitish tops; as red circular or irregular excoriations, or as granulating surfaces projecting as white moist raised growths. The mucous patches in the tongue are precisely like those seen in other mucous membranes and indeed are identical with them.

They may occur with the first onset of constitutional symptoms or not show themselves till a remoter period of syphilitic inoculation. They are very prone to reappear after their supposed cure.

When syphilis attacks the tongue as a local infiltration of gummous deposit, it does so usually long after the primary inoculation, even after twenty or more years.

It may do so as a single or more commonly as a multiple more or less rounded infiltration of the submucous or muscular

tissue of the tongue, and the swellings may be pea-like or nut-like; at first these swellings will be hard, but as time progresses changes will occur in them. If allowed to run their course they will enlarge and break down, open and discharge; if treated they may soften and be reabsorbed, or wither and dry up, the latter change being very rare.

When this affection is allowed to run its natural course, the swelling will enlarge and subsequently break up; the hard lump will increase and become softer, the soft parts covering it in will redden, inflame, and open either by an ulcerating or sloughing process; and when the contents of the lump have been discharged, either a ragged cavity will be left to granulate or a fissure to heal; the edges of the cavity or fissure being under all circumstances perpendicular and sharply cut, as shewn in fig. e, Plate 1.

The cavity, when the parts have opened by a sloughing process will be more or less ragged according to the amount of destruction of the tissue of the tongue, and it will present a surface which will vary according to the stage of the disease. When looked at during the period of sloughing, the dead tissue infiltrated with the *yellow* infiltrating material, of a wet wash-leather appearance, will readily be recognised; and when seen at a later period, the irregularly excavated cavity with sharply cut perpendicular uninfiltrated edges will generally enable the surgeon to diagnose the disease from the one for which it is often mistaken, a sloughing cancer. The common want of enlargement of the lymphatic glands in this specific affection of the tongue is another help to diagnosis. At a later period when repair has taken or is taking place, an irregular, yellow, white cicatrix (leucoma) will be seen, *vide* fig. d Pl. 1, and the tongue eventually will show marked evidence of the destructive processes of which it has been the seat. Wasting of some parts of the tongue, scarring of others, mixed up with irregular cicatricial tissue being the chief characteristics of a repaired syphilitic tongue.

In tongues that are brought rapidly under the influence of appropriate treatment, the changes that have now been described may be considerably modified. Thus the nodular infiltrated mass may soften, and the deposited material may be reabsorbed. The tongue itself will become supple and more

natural and a cure may take place,—a cure, however, which, in some cases, is attended with a wasting of the portion of tongue that was infiltrated, or a loss of the natural papillary tissue upon the surface of the tongue which corresponds to the seat of infiltration.

What, however, is of far greater importance to remember, is, that a tongue which has been the seat of syphilitic disease frequently becomes the subject of a cancer. The altered nutrition brought about by the irritation of the one affection, encourages the development of epithelial disease. A relapse of this affection after an apparent cure is also very common.

Treatment.—When the diagnosis of this many-faced disease has been made, the line of treatment to be adopted is not difficult to lay down, for there can be but little doubt that some mercurial medicine is the most certain drug to employ, where there are no indications against its use; and, on this being rejected or found wanting, the iodides of potassium, sodium, or ammonium, in gradually increasing doses.

The disease must be dealt with as a general and not as a local one; and the local affection is to be read as one of the manifestations of a constitutional disorder which has doubtless other seats which have not declared themselves.

When mercury is prescribed, the perchloride in doses of one-sixteenth of a grain in bark may be given; or what I like as well—a pill of half a grain of the green iodide of mercury twice a day. In both cases the dose being gradually raised to double the strength indicated.

When mercury is contraindicated on account of the patient's cachectic or feeble condition, the iodides may be ordered, commencing at 5 grain doses and steadily increasing them week by week by a grain up to 12, 15 or 20 grain doses three times a day. The iodide of sodium may be at times substituted for the iodide of potassium. Tonics are often required at the same time, with good simple food, fresh air, and regular habits. Stimulants should be given very sparingly and all smoking should be strictly prohibited. As a local application the lotion of boracic acid or chlorate of potash gr. x to the ℥j of water is of value, and the recommendation of Mr. H. Morris to rub a piece of blue-pill mass once or twice daily over the surface of the sore is worthy of adoption. When the disease has

apparently disappeared, the treatment must be continued for some, possibly for six months, this practice being necessary to guard against a relapse. The routine practice of applying the nitrate of silver to these sores cannot be too strongly condemned.

In the "lumpy tongue," in the stage in which the lumps are softening, I have found the simple operation of puncturing the tumours to be of great use—the punctures evacuating the contents of the lumps, which are often serous—thereby relieving tension and certainly expediting the cure.

In cases of long standing disease, the fear of the tongue becoming the seat of cancer should ever be before the surgeon, and the fact of a tongue having been the seat of an old syphilitic affection should tend rather to support than to weaken the view of a doubtful excavated ulcer of the tongue being of a cancerous nature. At any rate where the doubt exists let it rather encourage surgical interference than prolonged medicinal treatment, for in a clinical point of view a chronically affected syphilitic tongue had better be occasionally removed than a cancerous one left to run its course.

CANCER OF THE TONGUE.

This distressing disease is met with in about five out of every hundred cases of cancer, and is an affection of adult life; an analysis of 102 consecutive patients admitted into Guy's Hospital, and seen by me, showing that 80 out of every 100 affected by it were over the age of forty-five; 12 were under forty years of age; 27 between forty-one and fifty; 31 between fifty-one and sixty; 25 between sixty-one and seventy; and 7 over seventy years of age. This disease may, however, occur as early as twenty-seven. It is more common in male than female subjects, in the proportion of 80 to 22.

The disease is *always* of the epithelial form, and is essentially an isolated infiltration of the papillary or mucous surface. It usually shows itself as a blister, crack, ulcer, wart, or superficial tumour upon the tip or side of the tongue, and is in the majority of cases single. It then breaks down and discharges, (*vide* Plate II, fig. r) leaving a more or less ragged, irregular,

excavated sore with raised, indurated, infiltrated, and mostly everted edges (*vide* Plate II, fig. g).

The disease is at first always local, but later on, when allowed to take its course it will spread and involve the floor of the mouth, fauces, gums, or jaw bone. It will, moreover, always, sooner or later, implicate the lymphatic glands.

At times the diseased parts slough more or less extensively, (*vide* Plate II, fig. f), and in a case which was under my care in 1866, the whole organ sloughed off before the man died. It affects one side of the tongue as much as the other, and is at times central. But wherever it may commence, it will soon involve neighbouring parts.

It originates at times without any definite cause, but in the majority of cases it is excited by some local irritation such as that caused by a broken or rough tooth, a hot pipe, an antecedent syphilitic affection (Plate II, fig. f), or the disease which is now known as ichthyosis (Plate II, figs. g, h, i).

It may originate also in a scar on the tongue, as it is well known to do in scars of other parts. In 1880 I saw a case (Case 20) in which the disease had attacked the tongue of a man, æt. 57, who had bitten off its tip five months before in an epileptic fit, and a second in 1875, (Case 21), in a man, æt. 70, who had injured his tongue by a fall two years before.

Diagnosis.—Any localised *infiltration* of the papillary or mucous covering of the tongue, however limited it may be—in a patient over forty—should be suspected to have an epithelial origin, and should this be found in a part of the tongue in which no local source of irritation can be discovered, the suspicion becomes a certainty.

Should the infiltration coexist with ulceration and a local source of irritation be made out—such as a broken or rough tooth,—the probabilities of its being due to this local irritation may be regarded as great, but should the disease fail to undergo a rapid cure upon the removal of its supposed cause, the conclusion should be drawn that the disease is cancerous.

When a tongue has been the seat of a chronic syphilitic affection, and more particularly, is one in which a series of relapses has taken place, with uncertain intervals of apparent convalescence; and when it presents an indurated, infiltrated tissue, with a more or less excavated, ulcerating, or sloughing

cavity, with irregular, everted and raised, rather than sharply cut and defined edges, the diagnosis of the disease being cancerous is highly probable, and when with these symptoms the lymphatic glands beneath the jaw are found enlarged, the diagnosis becomes a certainty.

When again this local infiltration with or without ulceration is found in a tongue which has been the seat of an old syphilitic leucoma, or the subject of that peculiar disease of the papillary mucous membrane known as ichthyosis (*vide* Plate II, figs. c, H, I,), there should be no question as to its true nature, for it should be accepted as a fact that chronic syphilitic, as well as chronic ichthyotic disease renders the tongue peculiarly liable to undergo changes in its epithelial elements which most commonly reveal themselves as epithelial cancer.

Mr. Morris has recorded in an able paper on this subject ('Lancet,' May, 1882) the fact that out of fifty-five cases of cancer of the tongue, in thirteen, or about one-fourth, the organ had been the seat of ichthyosis. I am quite prepared to support him in this average.

Treatment.—There is but one form of treatment of cancer of the tongue that can be recommended with any confidence, and that is the removal of the disease by some surgical operation. And there is but one period at which this operation is likely to prove successful as a cure, and that is in the early stage of the disease, when the cancer is local and when it involves no other tissues than those in which it was primarily placed.

When the disease has extended beyond these limits and through the lymphatic channels has implicated the lymphatic glands, the prospects of a cure are not favourable even if they can be said to exist, since, whilst the glands that lie along the ramus of the jaw may be readily removed, those that lie buried behind the angle are beyond the surgeon's reach, and to remove some of, and not all, the infected glands is a futile proceeding.

When a local cancerous disease is removed it should be a rule of practice that all enlarged lymphatic glands should be removed likewise; and this rule is as applicable to the tongue as it is to other parts.

As to the best means for the removal of a tongue wholly or

in part—surgeons are found widely to differ—one advocating strongly the removal by the knife or scissors, whilst others as strongly urge the use of the *écraseur*, employed either as a crushing or as a burning force. The chain, or wire instrument, is used in the former case and the platinum wire heated by means of a galvanic battery in the latter. For many years I employed the galvanic *écraseur* and found no fault with it; of late I have again resorted to the chain or wire instrument, but have had no reason to be better satisfied with my results. I altered my practice in deference to a strong opinion that has been given by some surgeons as to the dangers of the galvanic and greater safety of the simple *écraseur*, but this opinion does not find support from facts.

With the view of testing this point I have extracted from our Guy's Hospital Records forty-six consecutive cases of operation, and find that of thirty-six operated on by the galvanic *écraseur* four died from the operation, or 11·1 per cent., and four from other causes. Of seven cases operated on by the chain or wire *écraseur*, one died from the operation and one from the disease. Of one removed by excision and two by ligature, none died. Of the whole number of forty-six cases five died from the operation, or 10·8 per cent., and five from other causes.

Of the five fatal cases from the operation, two sank on the 8th day, one from pleurisy and the other from broncho-pneumonia. One on the 20th day from broncho-pneumonia and one on the 20th from exhaustion and repeated small bleedings. The single fatal case after the use of the chain *écraseur* was on the 12th day from broncho-pneumonia.

One of the five cases that died after the operation, though not from it, sank on the 36th day from recurrent disease and gangrene of the lung. Three on the 53rd, 48th, and 32nd days respectively from recurrent disease and exhaustion, and one on the 38th day from recurrent disease and pyæmia.

It will be thus seen that *three* out of the eight cases that died after the use of the galvanic *écraseur*, and *one* out of two cases that had been operated on with the chain or wire instrument, or *four* out of the whole number of forty-six cases of operation, or 8·7 per cent, died from lung complication; and the records of the pathologist tell us that such a complication is

by no means infrequent when no operation has been performed. At any rate evidence is wanting to show that this lung complication is more common after operation than it is without, and that when it follows operation, such a measure has anything to do with the lung disease.

It is true that the inhalation of foetid or septic elements, when the tongue is sloughing either by natural processes or as a consequence of operation, must of necessity be prone to bring out this lung trouble; but this fact instead of being adduced as an argument against operative interference, may fairly be used as one in its favour, since to get rid of the sloughing and foetid organ is one of the surgeon's aims in an operation, and to do so in the quickest, safest, and simplest way is his object.

When the galvanic *écraseur* is used and the cauterised tissue is rendered aseptic by means of a plug of iodoform gauze well pressed upon the surface after operation; or when the charred or burned surface, after the use of the galvanic or wire *écraseur* is kept sweet by the repeated application of the colloid styptic which Mr. Morris tells us "tans the surface of the wound, causes little or no slough, and corrects the foetor of discharge," there is less fear of any evil result from septic causes than there was before the disease was removed.

There is consequently no argument against the use of the *écraseur* that has any weight.

Whilst, therefore, for the removal of a tongue, wholly or in part, I have a preference for the *écraseur*, and for the galvanic over the wire or chain instrument, I am ready to admit the value of excision by means of scissors or the knife, or of any of the different modifications of these operations which the ingenuity of different surgeons has suggested.

For I believe that in individual cases one form of operation may be more applicable than another, and that in the hands of any surgeon the mode of operating he excels in is the best for his patient.

I may say, however, that I have not yet seen a case in which the division of the lower jaw as taught by Syme has been required.

Most tongues can be removed through the mouth, however extensive the disease may be, if the organ be well

drawn forward by means of a thick ligature introduced through its body, and if it is freed from its attachment to the lower jaw and fauces by the division of its mucous membrane attachments. There is no objection to the removal of a whole tongue in halves, though there is no advantage in so doing. If more room should be required, this is best obtained by means of an incision across the cheek from the angle of the mouth, the *écraseur* being then worked sideways.

Bleeding during an operation need cause no alarm, if the operation be performed leisurely, since it can be speedily controlled by the torsion of the divided artery, if the tongue be well drawn forward; in many cases the simple drawing forward of the tongue suffices to bring about this result; the artery receding into the muscular tissue.

In the following case one of the objections to the use of the wire *écraseur* is illustrated:

CASE 22.—In 1867, Mary M—, æt. 44, came under my care with a cancer of one side of her tongue of one year's growth. I removed it by the wire *écraseur*, and in doing so stretched the lingual gustatory nerve. As a consequence the patient suffered for days from intense pain in all the parts supplied by the fifth nerve. The wound, however, healed well, and a good recovery took place. The disease however returned in the cervical glands within six months, and destroyed life in a year.

I can see no advantage in adopting the practice of Demarquay, of ligating before the operation the lingual arteries, although when severe bleeding takes place after the operation the practice may be good. The operation, however, may be performed, in cases in which the removal of the disease is inexpedient or impracticable, with the view of bringing about wasting of the diseased organ. The division of the lingual gustatory nerve on the inner side of the lower wisdom teeth for the purpose of relieving pain is also a practice to be recommended.

It must likewise be recorded as one of the advantages of the operation, that should a return of the disease take place it is more likely to do so in the lymphatic glands of the neck than anywhere else. Under these circumstances the patient is

relieved of his distressing local affection and sinks slowly and comparatively painlessly. I have often heard with pleasure, even under these miserable conditions, expressions of gratitude from patients who have gone through the operation; gratitude for the sufferings they have been saved from and spared.

I may also add that it seems probable that life is materially increased by the operation. In some cases I have to record, the increase was certain, and even when a return takes place it is so to a degree. Mr. Morris states that out of fifteen cases operated upon, the average duration of life was sixteen months, whereas in those in which no operation was performed it was but ten and a half. Only two cases having been known to have lived eighteen months.

In the cases I now record a decided increase to life must be admitted, and particularly if we take the average of life with this disease when left alone as ten and a half months.

CASE 23.—In 1866 I removed the anterior half of the tongue from W. P—, æt 60. The patient remained well so far as the tongue was concerned for *fifteen years* when disease reappeared in the scar. The patient at this time was suffering from hemiplegia and senile decay, of which he died.

CASE 24.—In 1872 I removed from Mr. S—, æt 45, half his tongue for a local cancer. He reported himself to me as well *ten years* later, 1882.

CASE 25.—In 1871 I removed a local cancer from the tongue of Mr. R. W—. No return ever took place though the patient lived *five years* and died from anæmic gangrene of the foot.

CASE 26.—In 1870 I operated on H. S—, æt. 70, and removed a cancer with the anterior two-thirds of the tongue. *Three and a half years* later he reported himself as well.

CASE 27.—In 1869 I removed a portion of the tongue from H. J—æet. 42. He lived *two years* and had no return and died from lung disease.

I can trace two cases now alive and well who have been operated on for *two years*, and two who are well one year

after operation, and have records of three who survived the operation for 18, 18, and 11 months respectively.

Altogether out of a somewhat limited personal experience the above record of facts must be regarded as encouraging, clearly showing the possible benefit of the operation, and the probability of a cure being obtained in a larger number of cases, if the operation were undertaken as soon as the diagnosis of the local disease has been made.

TUBERCULAR ULCERATION OF THE TONGUE.

It is right that this disease should have a special notice, since it is neither common nor generally recognised. It has, however, features of its own which claim attention. I have seen several examples of it but have only records of two.

It occurs in feeble subjects and begins as a papule which soon ulcerates and in spite of treatment passes into a sore or fissure. A second and third sore soon follow the first and run the same course, the same in its obstinacy and the same in its tendency to form a fissure or excavation. When it presents the latter appearance, the surface of the sore will be that of an old indolent ulcer on other parts; its base will be more or less infiltrated, but never hard like that of cancer; and its edges though infiltrated will not present the sharply cut aspect of the syphilitic fissure, nor the elevated everted irregular border of the cancerous; the secretion from the sore is often cheesy. In fact the sore is neither like the syphilitic nor the cancerous, and yet for want of its recognition it is usually taken for one or the other. The following case is one in point.

CASE 28. *Ulcer of tongue. Excision by wire écraseur.*—Annie C—, a delicate, strumous girl, æt. 18, was admitted into Lydia Ward on Feb. 19th, 1880, under Mr. Bryant's care. Two years ago patient noticed a little lump on the dorsum of her tongue about the size of a small pin's head. A medical man touched it with caustic but it has increased in size ever since. Nine months later, she noticed a small ulcerated spot at the under surface of the anterior portion of the *left* half of the tongue, and to this the same treatment was applied, but with no

benefit, the ulcer having been steadily increasing in size up to the time of her admission.

On admission.—On the centre of the dorsum of the tongue is a transverse fissure about half an inch in length and depth with somewhat infiltrated edges (Plate III, fig. 2). It looks as if a piece had been bitten out. In front of this is a small circular ulcer with a hard base and indolent surface. On the under part of the anterior portion of the tip of the tongue is a much larger ulcerating surface which is injected and indurated around the edges, and the infiltrating substance involves the whole thickness of the tongue (Plate III, fig. 2). Throat is healthy; manipulation does not give pain. There is one enlarged gland (sublingual) in the left side. The teeth with one exception are good; there is no history of syphilis.

On April 6th the mouth was opened by a gag, and three needles were passed through the tongue beneath the growth, in order to isolate it, the anterior half of the tongue was then removed with the wire *écraseur*. Three bleeding vessels were secured by torsion.

7th.—Temp. 98.4° to 99.2°

8th.—Temp. 98.6°

9th.—There is a large slough on stump of tongue. Has a great distaste for milk and cannot swallow solids, otherwise doing well. Temp. 98.4°

12th.—Tongue looks better and part of the slough has come away. Health improving.

14th.—Whole of slough came away.

15th.—Can eat without trouble.

16th.—Went out, convalescent.

Examination of the growth by Mr. C. J. Symonds.—The specimen shows the existence of a nuclear infiltration of the mucosa and deeper parts, with a destruction of epithelium. Plate IV, fig. 1, is a drawing showing the elements composing the nuclear growth. These are seen to be (a) small cells, for the most part rounded, containing many nuclei, and measuring about $\frac{1}{3000}$ of an inch in diameter. (b) A fine intercellular network in close connection with these cells, becoming coarse and fibrous at the periphery of a nodule. (c) Large multinucleated cells. These giant cells vary in size, that shown in the figure measures $\frac{1}{250}$ in. across. Others were found to

measure $\frac{1}{500} \times \frac{1}{666}$, and $\frac{1}{444} \times \frac{1}{500}$ inch. Many are smaller, all are irregular, and connected by fine processes with the trabeculæ. The nuclei are oval, contain several nucleoli, and congregate chiefly at the margin of the cell. In some of the cells the nuclei are placed at one end only.

The arrangement of these elements varies. The first morbid change seems to be the appearance of the small cells in the mucosa. For the most part they are diffused through the tissue irregularly, extending in all directions, separating the muscle fibres, as seen in fig. 2, like a sarcoma; the fibrous bundles are destroyed in like manner.

The epithelium over the growth becomes thinned by gradual removal from below. An increase of nuclei appears amongst the epithelial cells, resembling those of the growth described. As these accumulate the epithelium disappears, and gradually the small-celled infiltration reaches the surface; the corneal epithelium becomes confused and granular. It cracks and separates, leaving the cell growth exposed (fig. 3). This seems to be the way in which the ulcer formed. Besides this diffuse arrangement of the cell growth, there is another, and much more striking one. Rounded masses are seen mostly in the deeper parts of the section, and often isolated, containing in their centre large multinucleated cells, and bounded by a fairly distinct margin (fig. 4). Some of these nodules are visible by the aid of an ordinary pocket lens, three of them gave the following measurements:— $\frac{1}{33}$, $\frac{1}{50}$, $\frac{1}{100}$ of an inch. They consist mostly of the small-celled growth already described. For the most part they contain one or more multinucleated cells. Fig. 1 is taken from the periphery of the larger nodule in fig. 4. Towards the centre is seen a large multinucleated cell, connected with the fine intercellular network. Next comes the main component, the small-celled, "lymphoid" tissue, and finally, a denser and coarser network forming the boundary of the nodule. Beyond this is seen the muscle undergoing destruction, partly by the infiltration of the cells and partly by direct pressure. Fig. 2 is taken from the periphery of another nodule, and shows the muscular fibres curved by the pressure of the growth and undergoing atrophy. When containing more than one giant-cell, the nuclear growth of the nodule is often arranged concentrically around each,

giving the appearance of secondary nodules, and in some of the larger masses the circular arrangement exists without the giant-cell.

The disease begins in many independent points, which ultimately coalesce. Thus a rounded collection of the small cells occurs at one place in the mucosa, isolated from all the other growth, and surrounded by healthy tissue. This particular mass does not contain a giant-cell. At no place is caseation distinctly exhibited. Towards the surface, where the cells are abundant and where the outline of the nodules is obliterated, the staining is less definite, as if caseation was about to begin. As, however, I have not been able to get a section actually through the floor of the ulcer, *i.e.* as none of the sections are totally devoid of epithelium, it is possible the caseating parts have been missed.

The appearances, it will be observed, resemble very closely those seen in lupus, where there is also a small celled nuclear growth, with delicate intercellular tissue, and giant-cells. The lupus nodules, however, have not the same limitation (so far as I have seen) as have those in the present specimen, nor are the giant-cells so large. Moreover, in lupus, the giant-cells are scattered irregularly amongst the tissue, and do not form the centres of the nodules as seen here. Still, while there is much in which this specimen resembles lupus, there seems a greater weight in favour of its tubercular nature.

CASE 29.—*Tubercular ulceration of tongue; phthisis; death* (reported by Mr. L. H. Stevenson).—J. P—, a lithographer, æt. 50, was admitted into Job Ward on the 17th December, 1874, under the care of Mr. Bryant. The man had enjoyed good health with the exception of a slight rheumatic attack when twenty-eight years old. He was a married man, and father of ten children. One son having died of typhoid fever, the remainder were all living and healthy. His father died of asthma at the age of sixty-four, and his mother of apoplexy aged sixty-two.

Three years before admission a sore, which soon became a fissure, appeared on the left side of his tongue, for this he was unsuccessfully treated at a London hospital. Three months later, a second sore appeared, which was treated for twenty

weeks without benefit; he then went to another hospital for ten weeks where he got stronger, but his tongue resisted all treatment as it had done from the first.

When admitted his tongue was much swollen, and presented six or seven fissures chiefly on the left side. The fissures were through the tongue, and the tongue itself was infiltrated in the part. The edges of the sore were infiltrated, undermined and ragged, but neither sharply cut nor elevated and everted. He had a good deal of pain in eating, and for some time had taken nothing but liquid food; he also had considerable difficulty in speaking, appearing to suffer from a sort of spasm of the muscles of speech. The left side of his tongue presented a flat, perpendicular wall, instead of the usual edge, and the surface of the sore was very indolent; at the extremity was a small fissure, and another was situated about half an inch to the right of it.

He was ordered Ext. Cinchonæ gtt. xx, and a lotion of Soda and Borax.

January 6th.—The ulceration extends quite round the tip of the tongue, and is covered with patches of adherent lymph; the whole tongue looks livid.

Soon after admission the patient's lungs were examined, and on the right side some slight dulness and tubular breathing, as though from old mischief, were found, but no marked signs of active disease.

14th.—His tongue certainly seemed smaller, and appeared rather better.

15th.—Temp. 98·4°.

20th.—Temp. 102·5°, pulse 114.

21st.—He appeared very unwell, moaning continuously, and complaining of pain in his chest. A poultice was applied, but he died about eight the following morning. The post-mortem examination disclosed extensive phthisical mischief in both lungs. Lungs full of tubercles. Intestines ulcerated. Sections of the tongue exhibited numerous spots of tubercular deposit. The post-mortem was made by Dr. Fagge, but my colleague, Dr. Goodhart, who carefully examined this specimen, confirms Dr. Fagge's report in every particular and kindly reports as follows:

"I well remember the case of J. P—, from the fact

that, being asked to look at the tongue, I predicted the existence of phthisis, and yet, on examination, was disappointed at not finding certain evidence thereof. The case passed out of mind, until some weeks after the man died and his body was inspected. Then it was that the accuracy of the indications conveyed by the appearances of the tongue were verified. The disease of the organ in this case was extreme and therefore peculiar, its swelling and the consequent rounding of the edges exposing the sublingual mucous membranes might have suggested cancer or perhaps still more some syphilitic affection. But here, as in all cases, the character of the ulceration was quite distinctive. The ulcers were irregular, their edges but little thickened, or hardened, and their floors were formed by a soft-looking, cheesy material. Add to this, that the cuticular covering of the tongue was unnaturally glazed and red or livid, with numerous small superficial erosions of similar type to the larger ones affecting a large part of the surface, and the disease makes a picture which, once seen, is neither likely to be forgotten nor mistaken.

“Sections of the tongue showed that the cheesy material had invaded the muscular structures to some depth, and of the microscopical examination it is only necessary to say that it revealed a thick infiltration of the tissue by lymphoid cells, which were in many parts granular from degenerative changes.”

Altogether the clinical and pathological features of this disease as revealed in the reports of the two cases just recorded, make up a picture which possesses characters of its own sufficiently marked to render its diagnosis tolerably clear.

Could the ulcerating surface have been well scraped and thus destroyed, it is probable that a cure might have been brought about, but this method of treatment could hardly have been carried out in such an organ as the tongue; at any rate its removal answered well, as a speedy cure followed.

The case is allied to chronic inflammatory sores of other parts in which the inflammatory elements organise as granulation tissues, and dip down deeply into the parts around. In such nothing less than the complete excision or scraping of the infiltrated tissue will bring about a cure.

The following case is the best example of the kind I have seen :

CASE 30. *Sore on the right wrist with infiltrated edges; excision; recovery* (reported by Mr. E. O. GIBLIN).—Thomas S—, æt. 12, a fairly nourished boy, was admitted into Job Ward on the 13th November, 1872, under Mr. Bryant's care, having on the anterior surface of the right wrist-joint two open sores, one above and one below the anterior annular ligament; with a band of indurated raised skin between them. The margins of the sores were jagged, indurated, raised, and undermined, they contained yellow sloughing tissue, some black blood-clots and granulations. The skin around was healthy; a little to the outside between the two was the scab of a smaller sore. There was no pain except when the parts around were pressed. The boy had never had any previous illness. His mother, brothers and sisters were alive and well, but his father was always ailing.

Three years before admission he had a warty excrescence on his hand; this he pulled out; it bled very much and left a sore place, which has remained open ever since, and has spread an inch up the arm and an inch on the hand. He has been an out-patient at Guy's Hospital for three months, taking iodide of potassium and applying zinc ointment.

Nov. 14th.—A splint was applied to the hand and stimulating lotions to the sore, but without much benefit, indeed the ulcers spread, and on the 12th December the two had joined at their adjacent angles on the outer side; the sore was very painful, especially at night, and there was great thickening at the edges.

Dec. 16th.—The sores were washed with nitrate of silver lotion.

23rd.—Pulse 132. Temp. $102\cdot8^{\circ}$ The pain in his hand increased and his skin was hot and dry, he felt better, however, the following day and the pain in his hand was less.

31st.—The sore had not improved at all but looked less healthy than it did some weeks before. Chloroform was then administered, and the whole surface of the sore cauterised with the electric cautery, oiled lint was applied, and a metal splint was put on from elbow to fingers.

1873. January 8th.—Most of the cauterised surface of the wound has sloughed away; the granulations are very pale; but there is very little pain about the hand, and the boy's general health is very good.

11th.—There was a copious discharge of blood from the sore, the edges being still very indurated, and the surface irregular.

21st.—Tinct. Benzoin Co. was applied, and the sore was excluded from the air.

February 6th.—The sore was uncovered, the edges near the wrist were much more healthy; it is apparently healing.

14th.—The tissues over the back of the wrist-joint became swollen and the skin over it slightly inflamed, he was unable to move his wrist.

18th.—The boy was put under the influence of ether and an incision was made round the ulcer; the granulation tissue was dissected off the tendons; what was left behind had to be scraped away; some vessels were twisted.

21st.—The fingers were numb, he could feel over all except the palmar surface of the little finger. The sore was simply bandaged.

28th.—There was a yellowish-white discharge from the surface of the sore.

March 4th.—The sore was very much smaller, and looked very healthy.

On the 12th the sore had healed and he could move his wrist easily without pain; he left the hospital well on the 2nd of April, 1873.

On June 15th he was still quite well.

Dr. Goodhart kindly examined this specimen, and reported: "In examining the edge of this ulcer after its removal it was evident enough why it had not got well under less severe measures than the one ultimately adopted. The border showed nothing more than vascular and organising granulation tissue; the cellular growth had procured for itself a sufficient blood supply, and it was overgrowing even to the extent of gradual invasion of the parts around, and so the ulcer increased rather than diminished." The inflammatory growth was clearly growing as do new growths, and required a like treatment, viz. removal.

ICHTHYOSIS OF THE TONGUE.

This peculiar disease of the tongue, to which the attention of Surgeons was first drawn by Mr. Hulke in 1864 (Clin. Soc., 1868) is now generally recognised, although it is often known as psoriasis. It is met with in several forms. In the *least* common variety the papillæ themselves seem to be hypertrophied, and the disease appears as a coarse tongue in which the papillæ are very large, and, in some cases, covered with a dendritic, horny, epithelial covering (Plate III, fig. 3). In the *more* common kinds the surface of the tongue wholly or in part assumes, on the one hand, a smooth and bluish-white appearance, tessellated in a small or large pattern, and delicately furrowed, with an absence of papillæ (vide Plate II, fig. 6); or, on the other hand, it presents a more or less extensive whitish or yellow raised plaque made up of finer or coarser epithelial elements, with the uniform surface illustrated in Plate II, fig. 1, the parts when wet having a wet white-kid or yellow wash-leather aspect and a harsh feel, and when dry a brown appearance and a horny touch.

These three forms of disease may well be called, as suggested by Mr. Morris, the Papillomatous (Plate III, fig. 3);

Smooth tessellated (Plate II, figs. 6 and 8);

Raised plaque varieties (Plate II, fig. 1).

The disease is generally met with in subjects of middle age, although I have seen it in a woman as young as twenty-two, and it is more common in men than in women. It is in the majority of instances confined to the tongue, but in about one third of the cases the buccal membrane is implicated as well.

It is very frequently associated with cancer, as in four out of the last ten cases I have noted, and in thirteen out of twenty-seven tabulated by Mr. Morris; it is said by some authors always to lead up to it. It may or may not be found complicated with syphilis, but that it has a syphilitic origin can with some confidence be denied.

In one instance I found it associated with elephantiasis

arabum of the legs and genitals. It was in a gentleman, æt. 42, who had had the elephantiasis for ten years, and the ichthyosis of the horny type much longer. It is met with in the temperate, but more frequently in the reverse, and it is as often as not found in those who do not smoke. It is a slow, insidious disease, and is rarely recognised except by accident, until it has assumed a very marked type or become the seat of changes which suggest or characterise epithelioma.

The microscopical features of this disease are somewhat characteristic, they have been well described in the following report made by my friend, Mr. Symonds, of a specimen (Plate II, figs. G and H) which was taken from Mr. B., æt. 64, in 1882, in whom the ichthyotic disease had existed for twenty years, and the cancer for seven months.

"The microscopical examination shows the papillæ to be much wasted, their arrangement resembling more that in the skin than in the tongue.

"The superficial layer of epithelium is very much thickened, as shown in Plate V, fig. 1. The deeper layer varies in different parts. In the drawing the deep part is ill-defined, and the cells are seen with a higher power to become altered, and to be mingled with those of the mucosa. As the epithelioma is approached the limit is more marked for some distance, and the processes are shorter, until the epithelial ingrowths are reached.

"Towards the opposite side of the tongue the appearance resembles for some distance that indicated in the sketch. The mucosa is infiltrated with crowds of nuclei. These are particularly abundant at the spot from which the sketch was made, they diminish rapidly towards the epithelioma, but remain fairly abundant in the opposite direction. This diminution of nuclei, with increased definition of the basement membrane towards the epithelioma, is a striking feature in the sections."

Treatment.—It has been already stated that the majority of the cases of this disease do not come under the notice of the surgeon until the affection is a confirmed one, and under these circumstances it can readily be understood why the affection has been pronounced incurable. If seen earlier and

treated, there is some reason to hope that benefit might be derived from treatment if not a cure brought about.

This hope is supported by the assertion of those who tell us that they have seen cases of so-called psoriasis (not syphilitic) of the tongue cured, and I am sure that in several examples of ichthyosis I have found arsenic as an internal remedy, boracic acid or chlorate of potash as a local one, and as near an approach to milk diet as possible, with a total abstinence from wines, spirits and smoking, highly beneficial.

In cases of advanced disease it is difficult to find patients who will submit for a sufficient period to this treatment, but in others where it is met with in its early stage, the treatment would not be of necessity so prolonged. I am, however, convinced of its value, and would urge its adoption.

In confirmed disease I know of nothing but the excision of the organ, wholly or in part, that can offer any prospect of effecting a cure, and if the pathological doctrine be correct that this disease always ends in cancer, there can be no difficulty about the course which should be taken. I think, however, at present it may with some confidence be asserted that this positive opinion is "not proven," and under these circumstances whilst the surgeon is justified in not rushing into operative interference in all cases, he should so watch the case as to be prepared to take steps for the complete removal of the affected organ as soon as he can see that active changes are taking place in it, or anything like an ulcerative or degenerative change makes its appearance.

If the surgeon should err, let him do so on the side of early interference rather than that of delay, for it must be added that when a cancer attacks a tongue the subject of ichthyosis it usually developes rapidly, and when the disease returns after removal, it does so more commonly in the submaxillary or cervical glands than in the part. The return growth moreover always displays great malignancy. In the case from which fig. 1, Plate II was taken the disease returned within a year in the neck, and developed as a soft cancer.

In connection with this subject of ichthyosis of the tongue and its close association with cancer I should like to record the following fact.

CASE 31.—In 1879 I saw a gentleman, æt. 57, who was the subject of congenital ichthyosis of his skin. Had an ulcer on his foot of six years standing which became cancerous and had to be removed.

This gentleman was one of nine, the eight being women, and four of these eight had the same ichthyotic disease.

The mother of these nine had the same disease and her father before her.

The gentleman himself was married and had six children, three boys and three girls. Two of the boys were similarly affected.

CYSTIC TUMOUR OF THE TONGUE (BLOOD-CYST).

I give this case as it was reported with a drawing of the tongue. It seemed to be one of blood-cyst. I have seen nothing like it before nor since, and record it simply as a rare affection of the organ.

The correct features of the tumour are well shown in Plate III, fig. 4.

CASE 32. *Cystic tumour of tongue; incision into it, cure* (reported by Mr. E. Granger).—Frances C—, æt. 18, was admitted into Lydia Ward on the 21st July, 1875, with a fluctuating swelling at the back of her tongue, which came as far forward as the apex of the circumvallate papillæ. The mucous membrane over it was quite smooth; vessels well marked, large and distended; it seemed to extend back as far as the epiglottis. A peculiarity of speech had been noticed in the patient for many years, as also a peculiar movement of the tongue when taking food, particularly fluids; it was, however, only four or five months before admission that any swelling was noticed at the root of the tongue; it gradually and slowly increased, and ten days before admission she bled very much from the nose and mouth. Cold water was applied and the bleeding stopped.

Aug. 11th.—The cyst seems to have increased since her admission and the vessels over it are more distended and the speech less distinct.

25th.—The cyst was opened, and blood alone escaped; the cavity was plugged with lint. Microscopical examination of the cyst's contents showed only blood-corpuscles.

31st.—The swelling and fulness about the tongue has subsided and the anterior portion of the tongue presents a natural appearance. The cyst was gaping widely but was granulating from the bottom. It was still kept plugged with lint.

Sept. 23rd.—A chloral gargle was ordered.

Oct. 6th.—The cavity is filling up. There is still a swelling at the base of the tongue formed by the thick walls of the cyst.

11th.—She left the hospital.

Later on she came to report that she was quite well.

CASE 33.—*Mucous cyst on surface of tongue.*—Mary P—, æt. 41, came under my care on November 21st, 1864, with a growth the size of an almond on the left side of her tongue. It was fluctuating and translucent, and apparently contained mucus. I laid it freely open and evacuated its mucous contents, and a good cure followed.

This mucous cyst was precisely like those that occur in the lip, mouth, and in other parts of the tongue. When they occur far back they may affect the epiglottis, and as a consequence interfere with respiration. They are simply due to the obstruction of a mucous follicle.

TUMOUR OF TONGUE. CARCINOMA OR ADENOMA?

Other tumours of the tongue occur in practice, which cannot well be classified. The following is an example. If the case is looked at from its pathological aspect as shown by Dr. Goodhart's report, it must be regarded as a cancer of a rare form in the tongue, if it be not a gumma, but if we go by the clinical history of the case and its subsequent course after operation we must look upon it as a tumour of a simple nature.

The clinical history entirely excludes the probability of its having anything to do with syphilis, and the fact that six

years have passed without any signs of return, in a measure tells against the cancerous theory.

CASE 34.—Mrs. M—, æt. 37, the healthy mother of ten healthy children, consulted me on the advice of Dr. Wheeler, of Chelmsford, on January, 1876, for a tumour the size of a nut on the right side of her tongue. It had been growing for six months. It was clearly situated in the body of the muscles, and seemed to be a local infiltration, and on that account I took it for a cancer.

On January 29th I removed it by means of the galvanic *écraseur*, and the operation was quite bloodless; a good recovery ensued, and the lady is now well, six years after the operation.

I must add that the husband of the lady most positively denies having had any chancre or venereal disease.

I append Dr. Goodhart's report of the microscopical appearance of the growth, made soon after its removal.

"Dear Mr. Bryant,—You did well to excise that tumour *unless* there is any possibility of its being a *gumma*. It is not epithelioma and not glandular as I had thought possible from your description, but it has so much fibroid material in its structure, and the cells are of so little definiteness, that I incline to the belief that it is of a gummatous nature. But it infiltrates the muscular tissue of the tongue more than you supposed, so that if you will not allow its syphilitic nature you must consider it scirrhus and look out for a return. I can only give you one or the other alternative. Scirrhus of the tongue, though described, I have never seen, if this be scirrhus, before. Will you let me know if it might be due to syphilis?—Yours very truly, JAMES F. GOODHART."

CASE 35.—*Adenoma of the tongue*.—The following is an example of a rare form of tumour, which I removed by excision from beneath the tip of the tongue of Mr. L—, æt. 53, on February 2nd, 1881. The growth, when I operated, was about the size of half a nut, globular, and movable, beneath the mucous membrane. It had been growing slowly and painlessly for four years, and had given trouble solely from its position. The growth in its clinical features was not unlike the one last recorded.

After operation a rapid recovery took place, and Dr. Goodchild, of Little Waltham, Chelmsford, who sent the case to me, and has now charge of the patient, reports that "he is quite well and experiences no ill effects from the operation."

Examination of the growth by Mr. C. J. Symonds.—Two parts of a tumour, removed from the tongue of a gentleman by Mr. Bryant, were received:

1. A loose thin part, mostly membranous, and resembling a cyst wall; attached to which were several small nodules like part 2, and a little muscle.

2. A soft gelatinous piece, lobulated in parts, and translucent.

After hardening in chromic acid sections were made and stained with logwood (Plate VI). The structure appears that of an adenoma. The follicles are of various sizes, some when cut transversely covered the whole field, that is, looked as large as the whole of fig. 2.

All the follicles have a definite wall of fine fibrous tissue. In the best marked there is a regular lining of cells, and a finely granular material, apparently coagulated mucus, occupies the centre. In some parts the cell development is very abundant, little follicle wall and no stroma being visible. The stroma is a very fine fibrous tissue in the greater part. In some places scarcely any fibrous structure can be made out, the appearance resembling gelatinous tissue, but none of the cells characteristic of that material are visible.

CASE 36.—Aneurysm by anastomosis of right side of tongue.
—On July 16th, 1877, on the advice of Mr. R. Stevens, of Hoddesden, Herts, I was consulted by Mr. B—, æt. 30, for some affection of his tongue which he had discovered by chance one month before. It was the seat of no pain or inconvenience.

The case was clearly one of aneurysm by anastomosis of the arteries supplying the tip and right side of the anterior half of the tongue. This part was congested and swollen, it presented large veins full of blood on its surface, and large tortuous arteries were to be felt supplying it. The vessels

ran down to the base of the tongue. By pressure the whole spongy swelling could readily be emptied, and on its removal the growth refilled.

I wanted to remove the diseased part by excision, but could not get the patient's consent. No further history of the case can be discovered.

DESCRIPTION OF PLATES I—VI.

PLATE I. (*Coloured.*)

- FIG. A.—Hypertrophy of tongue.
- FIG. B.—Nævus undergoing cystic degeneration.
- FIG. C.—Undersurface of degenerating nævus of tongue.
- FIG. D.—Tongue, subject of old syphilitic disease.
- FIG. E.—Syphilitic fissure of tongue.

PLATE II. (*Coloured.*)

- FIG. F.—Cancer in tongue, seat of syphilitic disease.
- FIG. G.—Cancer in tongue, the seat of ichthyosis of the tessellated variety.
- FIG. H.—Section of cancerous and ichthyotic tongue.
- FIG. I.—Tongue, subject of ichthyosis of the raised plaque variety.

PLATE III.

- FIG. 1.—Tongue with tuberculous ulcer on surface.
- FIG. 2.—Tongue with tuberculous infiltration, &c.
- FIG. 3.—Tongue, seat of ichthyosis of the papillomatous kind.
- FIG. 4.—Blood cyst at root of tongue.

PLATE IV.

TUBERCULAR ULCER.

FIG. 1.—Taken from the periphery of a nodule to show the elements composing the nuclear growth.

- a.* Small-celled nuclear growth, with fine intercellular tissue.
- b.* Margin of nodule, showing condensation of the trabeculæ.
- c.* Giant-cell connected by its processes with the intercellular network.
- d.* Muscle undergoing atrophy. Hartnack, oc. 3, obj. 7 (reduced).

N.B.—The fibrous structure is too strongly marked, and a larger number of cells should have been shown. These were so numerous near the giant cell as almost to conceal the network.

PLATE IV (*continued*).

FIG. 2.—Muscular fibres from periphery of a nodule, showing infiltration by the nuclear growth, and wasting of muscle.

FIG. 3.—Showing the surface of the tongue.

- a.* Healthy epithelium, beneath which is seen the normal mucosa.
- b.* Epithelium becoming granular and broken. A rounded granular patch is seen to the left, resulting, probably, from degeneration of epithelium.
- c.* Infiltration of mucosa with small cells. The epithelium is seen to be thinned and to be undergoing destruction, especially on the right. The fibrous tissue in the deeper part is becoming replaced, and here multinucleated cells are seen.
- d.* Muscle undergoing atrophy.

N.B. to Fig. 3.—The degenerative changes in the epithelium, which lead to the formation of the ulcer, have not come out well in the plate.

FIG. 4.—Two nodules from the deeper part of the section. The larger shows the arrangement described in the text, and from this fig. 1 was drawn. Between the two nodules muscular fibres are seen much compressed and altered; while at other points bundles are cut transversely. Two nerves in section are seen between the nodules, and to the left an artery. These nodules were isolated from the rest of the growth, and appeared to be of independent formation. Figs. 3 and 4 represent the superficial and deep parts of the disease; between the two healthy muscle and connective tissue intervened.

Figs. 3 and 4 seen under a one inch objective, but reduced.

PLATE V.

ICHTHYOSIS AND EPITHELIOMA.

FIG. 1.—Taken from the centre of the section represented in fig. H, Plate II.

- a.* Corneal layer, much thickened.
- b.* Mucous layer, showing the deep cells mingling with those of the mucosa.
- c.* Mucosa crowded with nuclei.
- d.* Fibrous tissue of submucosa.
- e.* Muscle.

Hartnack, oc. 3, obj. 4 (reduced).

FIG. 2.—From the advancing edge of the epithelioma. Oc. 3, obj. 4.

PLATE VI.

ADENOMA.

FIG. 1.—A part showing the kind of tubes which made up the larger part of the growth. The stroma is shown surrounding, more or less, every follicle.

FIG. 2.—Selected to show the stroma, in some places fibrous and in others, as at *c*, almost homogeneous. At *b* is a large tube with a double row of cells, possibly representing a tube folded upon itself.

FIG. 3.—A large tube cut transversely, showing granular contents.

PLATE 1.

A



B



C



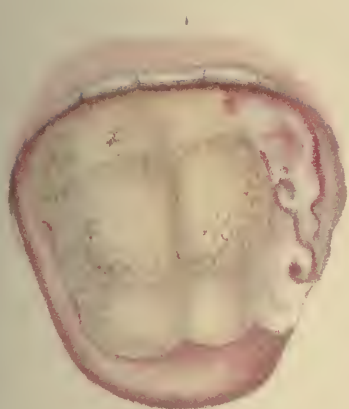
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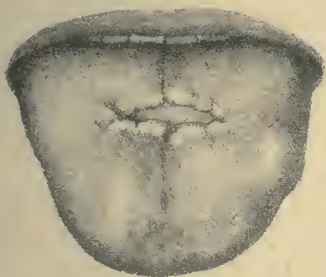
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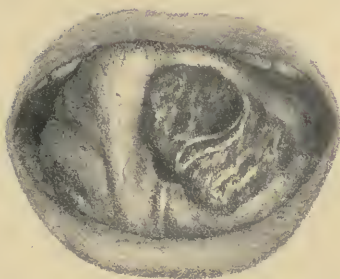
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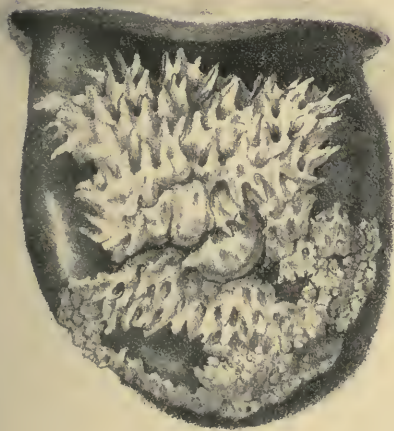
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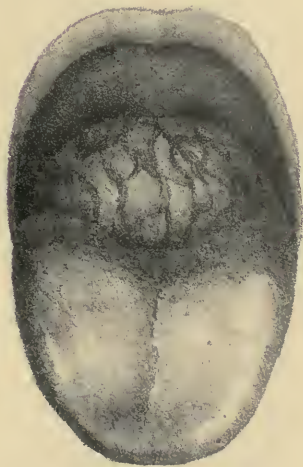
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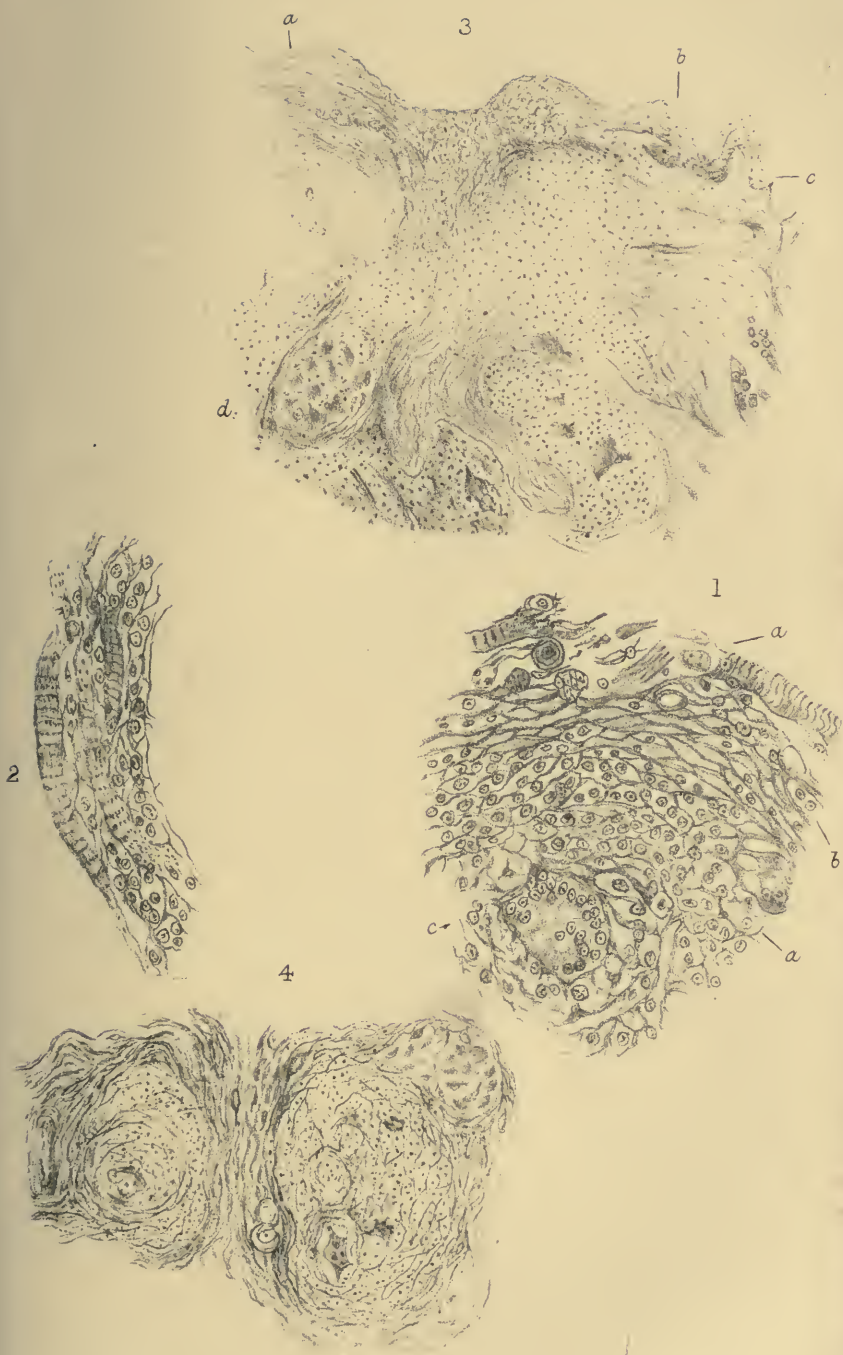


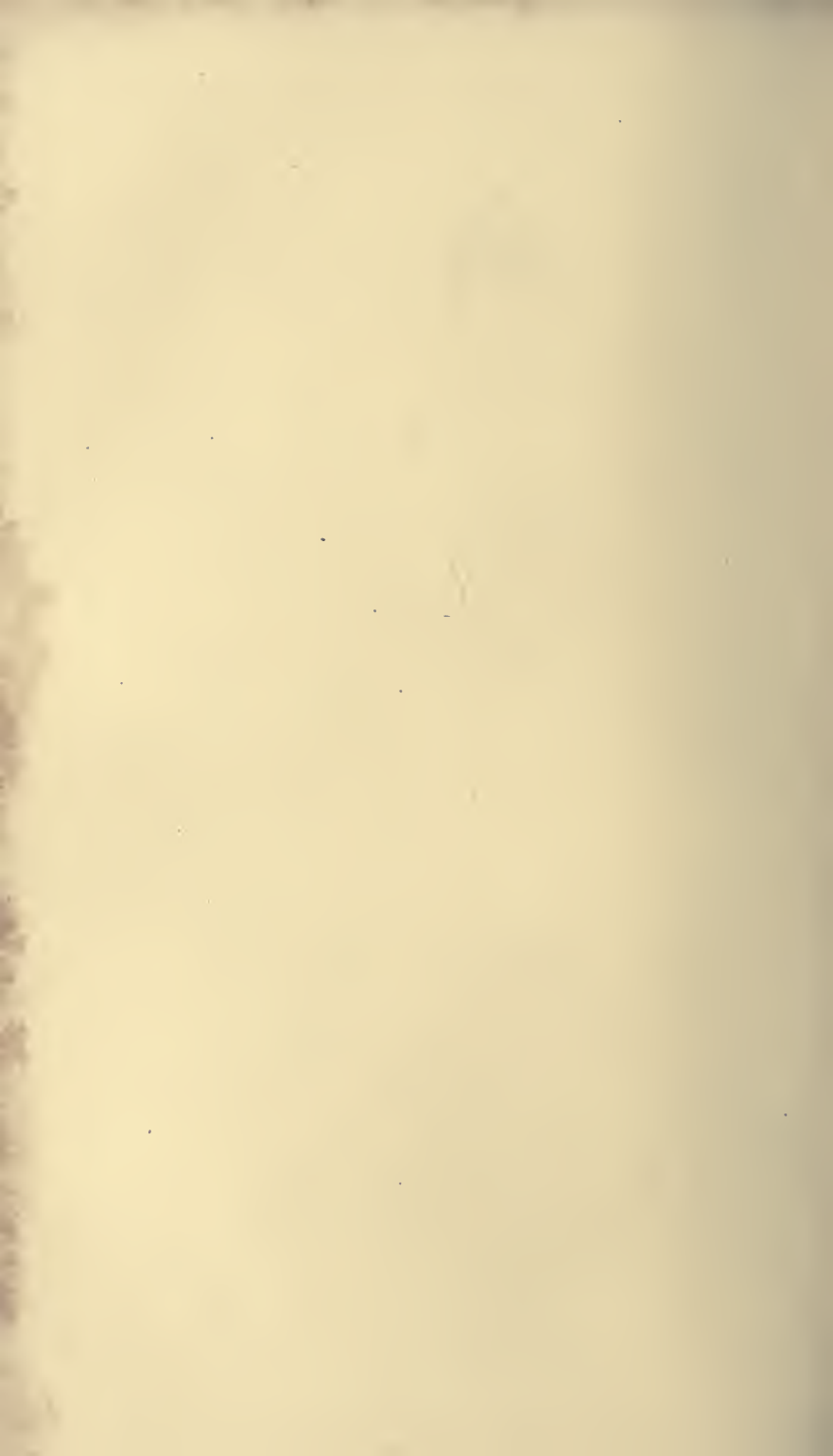
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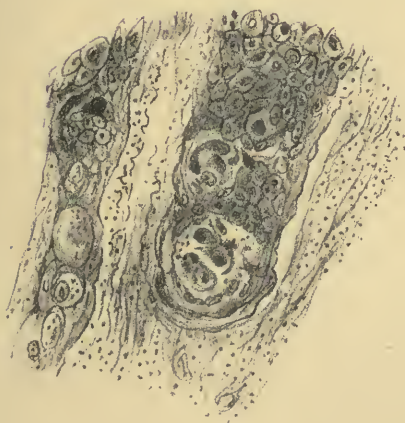


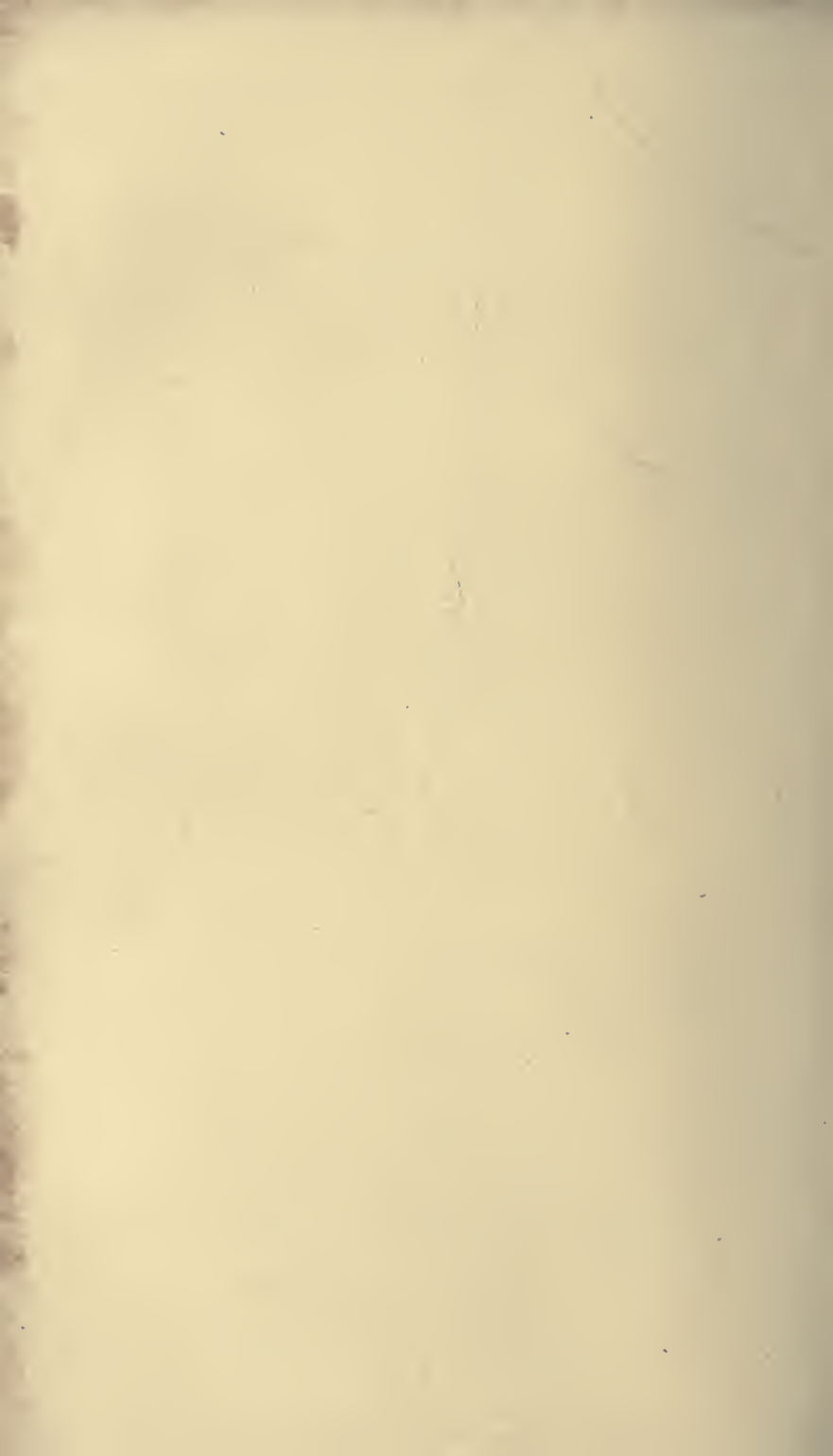


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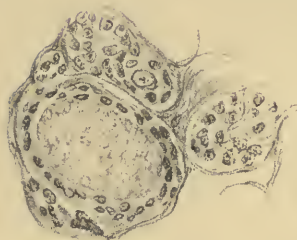


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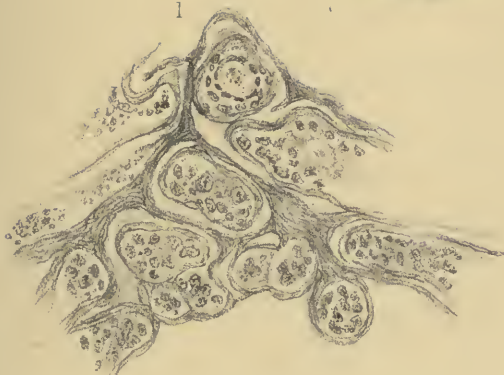




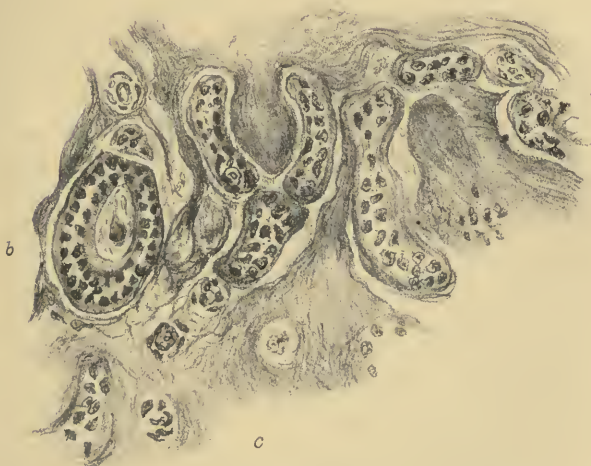
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ON HEMIANÆSTHESIA.

By SAMUEL WILKS.

HEMIANÆSTHESIA is a form of nervous affection which has of late created much interest both on account of the supposed discovery of its anatomical seat and also on account of the remarkable phenomena which attend its cure by the application of metals and other means. It is obvious that these two statements are somewhat antagonistic, and therefore the need of a further inquiry into the nature of the disease. The course which the sensory tract pursues, after passing through the pons and crus cerebri, has long been a question. Since the anterior part of the spinal cord is more associated with motion, and the posterior with sensation, it was long thought that the one tract, after leaving the crus, passed to the corpus striatum and anterior part of the brain, and that the posterior tract passed to the thalamus opticus and posterior part of the brain. This in a general sense is true, for disease in the region of the corpus striatum and in the cortex over it, is always productive of phenomena connected with motion and as a rule without any implication of the sense of feeling; whereas disease of the thalamus, although productive also of loss of motion, appears when certain parts of its circumference are involved to cause impairment of sensation at the same time. It seems that the sensory fibres are so blended with the motor that observers up to a recent period have failed to mark any spot in the brain which is exclusively their seat. Of late years, however, this discovery is supposed to have been made, and for the investigations

which have led to it, both pathological and physiological, we are indebted to Türck. According to his belief, the fibres of the cord which pass up from the crus containing motor and sensory tracts separate as they enter the brain, the motor being directed inwards between the two nuclei of the corpus striatum, and the posterior or sensory further outwards, between the lenticular ganglion and the thalamus. The whole of this tract is called the internal capsule; the anterior part is devoted to motion, and is that which is involved in hemiplegia, whilst the posterior part below the bend of the tract is sensory and is the region which is affected in hemianæsthesia. This doctrine seems to be now generally accepted, although apparently not in the same sense by all; for this small region called the posterior part of the internal capsule has according to many authorities a much wider function than the mere transmission of the sensation of touch, since it is supposed also to contain within it special sense fibres, and to be the seat of the lesion in those cases where the want of common sensation is associated with loss of all the other senses; that is, that combined with the sensory tract there are also fibres proceeding from the perceptive centres of all the senses running towards the convolutions. It will be seen from this that both from anatomical and physiological considerations the posterior or sensory part of the capsule is not analogous in function to the anterior. For the hemiplegia produced by diseases of the fibres in the corpus striatum is not a paralysis of half of the body but only of parts of it, whereas the hemianæsthesia produced by a lesion of the sensory fibres is in most cases not only a complete loss of sensation of the entire half of the body but of the special senses also.

I would not deny that it is in this region that the sensory fibres pass, and that there is such a thing as an anæsthesia owning an organic cause; but I am extremely doubtful as to the correctness of the theory which seems generally held, that the posterior part of the capsule has the wide function attributed to it. My own experience in reference to anæsthesia in connection with lesions of the brain is this, that in a large number of cases of limited effusion of blood in the brain, a hemiplegia results without loss of sensation. In others where the effusion is large and the apoplexy profound it is

impossible to ascertain the presence or absence of the sense of feeling, whilst in a third, and exceptional class of case, there is loss of sensation accompanying the hemiplegia. In cases of this kind the anæsthesia involves the arm and leg only, or if there has been any question as to its extension to the body the patient has been too profoundly affected by the brain lesion to render the diagnosis unequivocal. What I have not yet found and am in search of is a case of pure and simple hemianæsthesia due to a cerebral lesion. I believe I have met with cases where a lesion has produced a loss of sensation only, but then this has only been partial. In all cases where it has been complete the paralysis has been functional and, I believe, owned quite a different cause, as I shall presently attempt to show. In one of my cases, which I believe was organic, the patient was suddenly seized with loss of feeling in the arm and leg, and at the same time had sickness and other well-marked cerebral symptoms. After some months he was slowly recovering. The trunk was not affected nor the special senses. In the second case, the trunk was affected and the sense of touch was again the only one involved; the patient was so well in other respects that there was much doubt as to the existence of any organic lesion. The cases are as follows.

CASE 1.—A. B—, three months ago, whilst sitting indoors, suddenly felt a numbness in the left arm, and shortly afterwards in the left leg, and found a great difficulty in getting up to bed. These symptoms increased until he seemed to have lost the use of his left side. He lay on his pillow on his right side and found that any attempt at movement of the head brought on sickness. He continued in this state for a few days and then gradually recovered. He then went into the country and was able to walk some miles. He had not, however, gained complete sensation or power in the left limbs. He looked perfectly well and he walked as usual, but the grip of the left hand was scarcely equal to that of the right; it felt numbed, as if the fingers were in a glove; but yet when the arm was touched it seemed sensitive and a creeping feeling passed up it. He had no headache and no affection of the special senses. The heart was healthy as regards the sounds, but he had had rheumatic fever some years before.

CASE 2.—Mr. L. W—, æt. 38, whilst walking in his garden early in the morning a week ago, found the right side of his body had become numb, and this continued until he came to see me. This gentleman looked quite well, but on examining him I found that he had almost complete hemianæsthesia of the right side. On asking him to write he could grasp the pen to use it, but it would fall from his hand if he did not keep his eyes upon it; in walking, he said, if he did not continually look at his stick he should drop it. He moved the limbs awkwardly as if there was some amount of inco-ordination. The want of sensibility affected the body, face, and limbs, but the special senses were not involved. The skin and muscles were sensible to galvanism. He had no headache and no other symptoms whatever. I did not see him again.

In the first of these there was probably some lesion of the sensory tract, for the main symptoms were referable to the sense of touch only, and it is possible that the lesion might have been in the posterior part of the internal capsule. I do not doubt that the sensory fibres are to be found in this neighbourhood, for my own observations confirm the statement that where loss of sensation has accompanied hemiplegia the effusion of blood has extended backwards and outwards into the region spoken of. The same opinion is held by Dr. Crichton Browne, who found that in several cases of apoplexy and softening which occurred at Wakefield, the thalamus and its surroundings were more especially involved when loss of sensation accompanied loss of motion.

Bright had some belief that this was true, for he said if sensation was affected as well as motion, or if the sense of touch was more especially involved, that the lesion would be found posterior to the spot usually affected in hemiplegia. He gives the case of a young man, George O—, who had paralysis of motion and sensation, and in whom the former almost passed away leaving him partially anæsthetic. He died of pneumonia, and a softening was found at the back part of the corpus striatum. In another case, John W—, where anæsthesia accompanied hemiplegia, a softening was found in

the posterior and outer part of the corpus striatum near the junction of the cortical and medullary parts. In a third case, where similar symptoms existed, Dr. Bright formed the diagnosis that effusion of blood would be found in the left hemisphere of the cerebrum extending to the posterior part of the corpus striatum.

After admitting that there are facts to show that when sensation is involved in cases of lesions of the brain a particular region may be pretty confidently looked for as their seat, I now turn to the case of complete hemianæsthesia, associated with loss of the special senses, and inquire whether it be true that in this instance the cause is to be found in an organic lesion of any part of the brain, and more especially of the internal capsule. With reference to this point I stated some years ago that I had never met with an example of the kind, and that all my cases were functional and curable. Since this time I have been searching for examples which I could observe for myself or study from the records of others, but I have not yet been able to find a single case to disturb my former impressions.

I believe it is generally admitted that the large majority of cases of the kind which I mention occur in hysterical women, and it is these which have constituted the subjects of the interesting experiments of Charcot and his school. This form of hemianæsthesia implies a total loss of feeling of one side of the body, together with analgesia, so that pins may be thrust into the body without the patient being aware of it; at the same time there is loss of feeling in all the mucous membranes of that side, as the nose, mouth, conjunctiva, rectum, &c. With this loss of common sensation the function of the other senses has departed on the same side; the patient has almost lost the sense of hearing, and taste has disappeared so that salines and bitters placed on one side of the tongue can no longer be appreciated; smell likewise has gone, so that strong odours and stimulants are not perceived in one nostril; sight is also affected. Hysterical amaurosis has long been known, but Charcot has found that the eye may retain its power of discerning form whilst it has lost all perception of colour, the colour departing as a rule in a given series and returning in the inverse order. This achromatopsia is a fre-

quent accompaniment of hemianæsthesia. As occasional complications we meet with contraction of the limbs and epileptic fits constituting the complaint known as hysteropilepsy. It is worthy of note, also, that in many of these patients there is a perversion of their mental and moral faculties; symptoms not to be overlooked in considering the nature of the complaint.

The interest which this class of case has excited has not been so much owing to the supposed discovery of the local cause in the brain through the observations of Türck as it is to the remarkable effects produced on the patients by the application of metals at the Hospital of Salpêtrière. The method has been in a word called "metallotherapia." It was found that if metals were placed on the anæsthetic arm sensibility would return and that the susceptibility to different metals varied with the individual; and more than this, when the most appropriate metal was found, that if this were given internally, it would effectually cure the patient. The experiments were made with gold, silver, lead, tin, &c. It was also noted that when, after a certain time, say half an hour, sensation was restored, anæsthesia would appear on the corresponding part of the other arm; this was called the law of transference. It was subsequently found that similar effects might be produced by galvanism, magnets, or solenoids. The experiments being repeated in this country and on the Continent were verified in most particulars. When it was found, however, that discs of wood would produce the same effects, and that various other means were equally powerful, a scepticism grew up in England with reference to the supposed special influence of metals, and an explanation of it was founded on what is called "expectant attention."

I had previously given in these 'Reports' cases of hemianæsthesia with loss of special sense as instances of a functional affection, and now I add another to illustrate the treatment by the local application of metals. The case was taken by Dr. Horrocks, then house-physician.

CASE 3.—C. R—, a married woman, had been ailing since the birth of her first child a few months before. The child was stillborn, and she had some inflammation of the womb

after her confinement. She seemed an hysterical woman, and for the sake of testing her a needle was run into the left side of her neck, then into her scalp, loins, &c., but she was quite unaware of it. When pricked on the right side she immediately exclaimed. It appeared, indeed, that although she could feel objects, that she was totally analgesic on the left side. There was slight deafness on the left side; she could see forms with her left eye, but could not distinguish colour; she called all colours by their wrong names. She could not distinguish heat from cold on any part of the left half of the body.

Two sovereigns were fastened on her left forearm; after twelve minutes she felt the prick of a needle just below the coins. After sixteen minutes she felt the needle nearer the wrist, and there was some analgesia on the corresponding side of the right arm. The experiment was continued until she could feel the prick of the needle in other parts with a corresponding impairment on the opposite side. After a time she returned to her original state, and it was found also that she was quite anæsthetic. On the next day two discs of lead were applied, when after some time she could feel a little. Then two iron discs were applied, when sensation returned with a corresponding loss on the opposite side. Subsequently gold was again tried, and its effect in restoring sensation being more marked than that of the other metals, it was determined to give it internally, and one eighth of a grain of chloride of gold and sodium was given three times a day. At this time sensation was returning in the body, although still absent in the limbs. One night, on going round the wards, the house physician found her asleep, and pricked her left hand with a needle; she rubbed it, took it away, and finally, on the irritation being repeated, she awoke. She was then pricked again, and could not feel at all. After taking the medicine for a month it was found she could feel an ordinary touch and the prick of a needle down to the elbow; the anæsthesia and the analgesia were disappearing from above downwards. Up to this time she could scarcely walk; after another two weeks she could walk a little. She was then promised the pleasure of attending a concert in the hospital if she would try to walk; she improved so much that she could soon walk upstairs and

downstairs by herself. She was then sent home. She had still some anæsthesia and analgesia from the wrist down the fingers and from the ankle to the toes. Elsewhere sensation had returned, and she had regained the use of all the special senses. She took the chloride of gold for nearly eight weeks, but she was not aware of the nature of the remedy.

The details of this case render it impossible to dispute the facts which have been observed in connection with the cure of anæsthesia by metals, but I have grave doubts as to their *modus operandi*, as I have to the correctness of the theory as to the supposed seat of the disease. As so many instances have occurred in hysterical women I have sought to eliminate this difficulty by studying the affection in men, but I have been surprised to find that in many instances hysterical phenomena have also been present in them. This leads me to the belief that the latter are in some way essentially a part of the disorder, and must not be put aside in the consideration of its nature. I have, in a former number of these 'Reports,' spoken of the arrest of the function of the brain, as a whole or in part, as a means of accounting for certain temporary nervous phenomena. If I am right we have only to suppose a cessation of action of half of the brain or a part of it, say the middle and posterior lobes, to account for loss of perception of all kinds, just as we may suppose an implication of the anterior part to result in a loss of power.

A total loss of both power and feeling would imply a cessation of the action of the whole hemisphere of one side, and would carry with it also a complete aphasia. As an hypothesis this is more simple than the supposition that there exists a small spot in the brain whereunto converge the fibres of all the senses, and moreover that this spot should be picked out for such a lesion as a small effusion of blood, especially, too, as it is not shown to have any distinctive vascular supply. The facts are also on the side of the first theory, since the larger number of cases of hemianæsthesia recover, and in others of a permanent character, when subsequently a post-mortem examination has been practicable, no lesion has been discovered. At least this is correct as far as my own investigations have gone. Moreover, the general disturbance of the mental and moral

faculties would suggest the existence of a more universal affection of the brain than one limited to a few of its white fibres.

The reason why one hemisphere should cease to functionise may in many cases be difficult to ascertain, but in very many instances it is to be found in a physical or moral shock. There is no difficulty in accepting the fact that a shock of either kind will produce the symptoms under consideration in so extensive a way as to affect the whole brain and body, that is, by a total loss of power of movement, total anæsthesia, and a complete abolition of all the special senses. A blow on the head will render a person for the time apparently lifeless, in which condition perception has completely gone; even, indeed, when consciousness is returning the apathy may be still great. A moral shock will produce exactly the same phenomena. In my former paper I described the case of a girl who, owing to a sudden fright, fell into a state of lethargy, in which she lost the power of feeling as well as seeing or hearing. Now, we have only to halve these phenomena and consider that one hemisphere has received the physical concussion or moral shock to understand the case of hemianæsthesia and the theory on which it depends. There is one difference, however, in the cases of the greater and lesser shock, namely, the loss of consciousness in the former and its presence in the latter; but this is only what would have been expected. It is well known that consciousness may remain as well as volition when a very large part of one hemisphere is destroyed. When, for example, one side of the body is perfectly helpless from disease of one side of the brain, the other half of the body is still under the control of its supervising hemisphere in voluntary acts. There are many persons who from infancy have had a wasted hemisphere and a correspondingly withered half of the body, and have yet retained their individuality and their power over the healthy half.

The theory of the cessation of brain action may be unacceptable to some because it is novel, but a moment's consideration will show that probably very similar states of quietude or utter cessation of functionising may occur in other organs. There is, for example, the brain itself during sleep, when its activity ceases for a time; then we may note

also the intermittent action of the stomach, and the difference which must exist between a large organ full of blood, secreting gastric juice, and exciting a powerful influence on the food, and a small bag, empty, containing no active secretion, with a scanty flow of blood through its vessels, and to all intents and purposes inert. - We might also contrast the active and passive mammary glands, the testes, the lachrymal and other glands, whose function may for a time be almost dormant and at another be going on in great excess. Even other and more important organs, such as the liver and kidneys, no doubt undergo great differences in their activities during day and night. The influence of shock, both physical and mental, on the action of the various organs of the body has been often observed; there is not an organ of the body, whose secretion may not be affected by a mental emotion, and I have seen very remarkable temporary effects produced by a direct injury. With reference to the proposition that an organ like the brain may be structurally healthy and yet not in a state of functional activity, we may be reminded that if the brain be regarded as the organ of the mind the statement is evidently true, for supposing an uncultivated savage be taken, and become not only civilised, but highly educated, we are witnessing the case of an organ actively at work, which was before dormant.

I think it is manifest that the organs need not always be in a state of activity, but at times cease to functionise, and therefore the only proposition I ask the reader to accept is, that half the brain may be dormant, whilst the other is active. In confirmation of the reasonableness of this view, we may look for a moment at the case of hysterical hemiplegia and contrast it with the ordinary form due to a lesion in the motor tract. In the latter the conducting line is cut between the cortex and certain parts, such as the limbs and a portion of the face; the patient's will is good to move the limbs as seen by the effort made when partial recovery is taking place, whereas in the hysterical patient the fault lies in the will, and therefore those parts over which we have control are those which are implicated, the patient not only cannot move her arm and leg on one side, but she cannot open her mouth or raise the eyelid or contract her bladder. The case is, therefore, totally

different from the true hemiplegia and is not explicable on the hypothesis of the existence of any local lesion, but only on that of the will being at fault, that is, the higher centres of the brain contained in the cortex of the hemisphere. In this case every one admits that the will is at fault, that is, the will connected with one hemisphere only, the other will connected with the other hemisphere being still active.

If then we remember that one hemisphere may be disorganised with a corresponding paralysis, and yet the consciousness and will remain with ability to move the other side of the body; and if we remember also the case just mentioned of hysterical hemiplegia where those parts are paralysed which are directly under the influence of the will, and therefore that the latter must be in abeyance on that side, we cannot but conclude that functionally as well as organically one hemisphere may for a time be dead or inert. In this way total want of perception on one side of the body is easily accounted for. But I have said that mental and moral perversions were often present, and that they cannot be ignored in considering this remarkable affection. Now, it must be admitted that these aberrations would be more likely to occur in connection with a general disturbance of the hemisphere than with a mere local lesion in white connecting fibres. This, therefore, is another argument in favour of my theory. If the consciousness and volition may remain in each hemisphere separately, we must suppose that under normal conditions the two act in unison by means of the commissures, or, at all events, that there is some agreement between them, as when, for example, one hemisphere influences the bow and the other hemisphere the strings in playing the violin. If the two hemispheres did not act in agreement then there would be a temporary mental aberration; this has long been conjectured by many alienists, but is a subject too extensive to engage our attention now. It would, however, carry with it a moral aberration also, and therefore we see in this fact a further argument in favour of the hypothesis that the hemisphere is at fault in hemianæsthesia as it is in hysterical hemiplegia, and that just as its want of action produces an obliquity of the body, its inactivity causes also an obliquity of the mind.

An unequal activity of the two sides of the brain wherever

there is an unequal proportion between them, has long been considered a cause for mental aberrations; for it has often been observed, as Benedict remarks, that a want of symmetry on the two sides of the cranium is very common in both criminals and lunatics. The theory of the intermittent cessation of activity on one side of the brain, as we witness in the case of common sensation coming and going, will account for mental phenomena otherwise inexplicable; for example, the 'London Medical Record' for 1878 contained an account of a case by Dr. Berthier, entitled "A complex and exceptional neurosis." The patient was a young girl, æt. 13, to whom he was called. He found that she had gastric symptoms with delirium, and other nervous phenomena. He paid her several visits, and found that sometimes she could see him and at other times appeared blind, sometimes she heard at other times was deaf; one side of her body was anæsthetic and the other over-sensitive. Sometimes she was dull, timid, and not very intelligent, at other times very conversant and self-possessed. At times she would suddenly cry out and ask where she was as if awakened from a dream. This was after acting as if in a state of somnambulism. The transition from these states to that of normal life was very rapid and could not be predicted by more than a few minutes. I think this case is only an extreme example of what we constantly witness in hysterical women with hemianæsthesia. If we suppose the anæsthesia to be constantly coming and going as the nervous power oscillates, and that it be due according to my theory to constantly changing activities of the brain, we can explain the meaning of the phenomena in Dr. Berthier's patient.

It is unnecessary to insist on the perverted moral nature of hysterical girls in whom a right guiding principle so often appears to have been lost, but it must be carefully noted that similar moral obliquities have been apparent in many cases of hemianæsthesia, and, what is remarkable, not only in cases of women but in those of men also. The fact has constituted a very great difficulty in testing the accuracy of the statements made by these patients. Westphal, in experimenting on German girls in order to satisfy himself of the accuracy of Charcot's statements as to the effects of metals, declares that

they appeared to be true, but it was remarkable that every one of his patients had been in contact with the criminal law. In venturing my own opinion on the matter I have no hesitation in saying that having long thought that one of the most important physiological facts as regards our organisation is that the body is made up of two halves with a double set of senses, so I have considered that many strange ailments may be due to the irregular or unequal inactivity of the two sides of the brain, and that those who advocate the theory of mental and moral perversions being due to the same cause have much to show in its favour.

Now, if it be admitted that in hysterical and other persons one hemisphere may sink into a state of inactivity, it is another question whether a sudden physical or mental shock can produce the same. Seeing that exactly the same phenomena ensue from an injury as are seen in the case where I suppose one hemisphere has ceased to work, I necessarily believe that a blow on the head or a shake may produce a similar effect, that is, an arrest of the action of one hemisphere.

This is a most important point with reference to the question of cortical hemiplegia, for usually if paralysis of any kind or disturbance of the motor or sensory system follow a blow on the head a cortical lesion is surmised to have occurred, whereas, as another supposition, the whole hemisphere might have undergone concussion. The question asked is, Can a blow on the head cause a stunning of one hemisphere or a part of the brain, in the same way as it may produce concussion of the whole? The shock need not be of the same severe nature as a general concussion, so as at once to arrest the cerebral function, but it may be sufficient to start a process of an inhibitory kind which shall spread to the whole hemisphere. That injuries to the head may be the exciting causes of forms of paralysis exactly simulating the hysterical, I constantly see, and therefore I cannot under these circumstances believe that the injury has produced any local lesion, but has merely disturbed the brain as a whole or in part. Although it is now many years since Sir. B. Brodie so lucidly unfolded the subject of hysteria in connection with injury, I yet find the general tendency is to connect the injury

with the subsequent symptoms in a too definite manner. Sir B. Brodie showed that the symptoms he described were not due directly to the injury but to the general nervous disturbance caused by it. As the subject is important, I will quote from his work on 'Local Nervous Affections.'

"In a great number of instances local hysterical symptoms appear to be connected with some accidental injury, generally a very slight one, and they are then especially liable to be misunderstood and mistaken for something very different from what they really are. For example, a woman is bled in the arm, she complains of pain extending down the forearm to the hand, up the arm to the axilla and shoulder, or to the neck. You examine the cicatrix and discover nothing unusual in it, but the patient flinches when it is touched. In another case the patient has received a blow on the head, she subsequently complains of pain in it and many other sensations referable to the seat of injury. In another case, a young woman pricks her finger or perhaps the finger is merely pinched, soon afterwards she complains of pain extending from the finger upwards along the hand and forearm. This probably is followed by a convulsive action of the muscles of the arm or by a continued contraction of the flexor muscles on the anterior part of the arm, so that the forearm is kept permanently bent, at least while the patient is awake, for the spasm is generally relaxed during sleep."

"A young lady, eleven or twelve years of age, pricked the forefinger of her left hand with the point of a pair of scissors. This was immediately followed by pain in the course of the median nerve, and on the following day the forearm was fixed by muscular contraction at a right angle with the arm. After a few days all the muscles of the hand and forearm were affected with violent spasms, producing strange convulsive movements of the hand and forearm. These were attended with sickness and vomiting. By degrees the other limbs became affected, and it was impossible for the patient to walk or even to stand. Sometimes the diaphragm was affected so as almost to threaten suffocation. At other times the jaw was closed by a contraction of the masseter muscle or she lay in a state of opisthotonos."

I could relate several cases where the affection of which I

am now treating arose after an injury and consequently a direct connection was thought to exist between the symptoms and some supposed nerve lesion. The complete recovery as well as the absence of all disease, when in a few instances a post-mortem examination was subsequently made, negatived this idea. Thus, some time ago, a patient was sent into the hospital by Mr. William Toulmin, of Clapton, for paralysis following an injury. She had fallen down over a bottle and had injured her back. She was soon afterwards found to have complete hemianæsthesia of the right side together with loss of sight, hearing, &c. She responded to the action of metals as has been described and made a rapid recovery. I am informed of a case of a young lady, which may come before the legal tribunals, where, after a fall, all the extraordinary phenomena associated with hemianæsthesia appeared, and where the same susceptibility to the action of metals was found. Skey describes the case of a young woman who sustained a railway shock, and had in consequence a total loss of sensation and motion on the left side and a partial loss of both on the right. There was excessive tenderness over the upper lumbar vertebræ. The loss of sensibility was so complete that she was quite unconscious of the prick of a needle. The paralysis was referred to a local lesion and disorganisation of the cord and its membranes. She completely recovered.

Dr. Donald Fraser relates in 'Brain' the case of an idiot boy who fell on his head, and the accident was followed by right hemiplegia and hemianæsthesia. He died after some days and there were found congenital irregularities but no lesion of any kind in the region of the left optic thalamus and internal capsule. Dr. Fraser says: "But for the opportunity of making a post-mortem examination the symptoms would have been considered as indicating plainly some such organic lesion as a considerable effusion of blood affecting the left hemisphere; while as it was, the most careful examination failed to show any gross lesion."

I will now relate a case where the usual symptoms of hemianæsthesia followed the receipt of an injury and were therefore thought to be dependent upon it.

CASE 4.—Eliza T—, æt. 49, was admitted December 11th, 1878, into Guy's Hospital, and the case was reported by Mr. R. J. Scott. She was then getting better from a right-sided paralysis. She was a little woman, hump-backed from spinal curvature, and was a dressmaker by trade. She gave an account of her illness as follows:—"Ten months before, she was thrown out of a cart and was very much shaken. About a week afterwards she found that she could not move her right arm or leg, and the right eye was so dim that she could not distinguish objects with it. She stated that her face was drawn up, and her speech was affected in such a way that she forgot the names of objects or called them by their wrong names. She remembered, too, that her taste was much impaired, although it was not specially tested. She also lost feeling in the arm and leg and could not feel when the face was touched. This state of things continued some months when her speech returned and she was able to move her leg and arm. Sensation also was returning, but she could not distinguish between heat and cold; at the same time a prickling sensation came over the face and right arm and leg. She had no paralysis of the bladder or rectum." On admission her speech was good, her sight had returned, but hearing was not so good on the right side as the left. The right arm had not so much power as the left, and the right leg was also weak. She thought during the time she was ill that the right limbs became smaller than the others and were now growing larger again. Sensation was very much impaired on the right side, so that she could not distinguish degrees of temperature and was much confused as to the shape of objects placed on the right arm or leg. She afterwards made a more rapid progress and was able to leave her bed about a month after admission and walk with assistance. After another month she was nearly well, walked in the ward, could use her needle, and the functions of the special senses had returned. The only symptom left was a feeling of pins and needles in the right arm and leg.

In former times, more frequently, perhaps, than at present, nerve symptoms were associated with supposed lesions; at least, I judge so from the perusal of Mr. Guthrie's work on

‘Injuries to the Head.’ I quote one of his cases which he trephined, but I have little doubt the patient might have been cured equally well by metallotherapy.

CASE 5.—M. A. T—, a stout, healthy-looking girl, received a blow two years ago from a stone falling from a doorway under which she was passing, which struck her upon the left side of the head at a spot an inch anterior to the parietal prominence. The immediate effect of the blow was insensibility, followed by acute pain in the head, which ever since continued to mark the seat of injury. A week after the receipt of the blow she began to lose the power of moving the right arm, there being, however, no loss of sensation or any disturbance of the cerebral functions. During the following twelve months the symptoms remained unchanged, and she visited several hospitals without relief. The paralysis then became more complete, and she was finally admitted into the Westminster Hospital. The arm and leg were then quite paralysed, the arm which had been flaccid was rigid, and vision and hearing were slightly affected. Mr. Guthrie removed a disc of bone from the exact point in the parietal region to which she referred the pain. The portion of bone showed no disease. An hour afterwards she raised the paralysed arm, and was able to extend the fingers, the pain was relieved, the countenance was less dull, and sensation was returning. During the following three days fever and rigors appeared as if inflammation of the brain was supervening. When these symptoms had passed the paralysis had completely disappeared, and the sight and hearing were regained. Mr. Guthrie adds :—“She afterwards had some relapse of pain and uneasiness in the head, but is altogether a different person, although of a very hysterical temperament. The cicatrix on the head is firm, and she considers herself to have been cured by the operation, although I find it difficult to say in what manner it was effected or why the removal of the bone, which was in a perfectly natural state, should have given relief.”

The loss of feeling, sight, and hearing sufficiently indicates the nature of this case, which is not explicable on the supposition of a local lesion. It is probable that in many other instances where a severe injury has been followed by

nerve symptoms, the latter have not been due to a local lesion but to a concussion, and thus an explanation afforded of their rapid subsidence. The very next case which Guthrie gives of trephining the skull in a soldier, who received a bullet wound at Waterloo, may have been of this nature.

I will now refer to cases in the male which tend very much to corroborate the opinion that hemianæsthesia is a functional disorder. I have been so well satisfied on this point that when I have found the existence of this condition I have at once pronounced upon its functional and curable nature, and have put on one side the previous verdict of a profound local lesion. I have not yet been mistaken. It will be observed that the male patients have been affected by the same kind of mental and moral perversity as exists in women, a fact of the utmost importance in attempting to form a conclusion as to the true nature of the affection under discussion. The facts seem to show that if from any shock, moral or physical, one hemisphere should be arrested in its action both physical and psychical consequences ensue. The cases first described and those which follow are too much alike to allow me any hesitation in expressing this opinion.

The following was sent to the hospital as a very severe case of paralysis due to apoplexy. So it appeared at first sight, but a careful investigation of the symptoms showed me its triviality.

CASE 6.—Wm. P—, æt. 19, a footman, was seized a few days before admission with pain in the head, giddiness, and sickness. He was put to bed, where he remained until he came to the hospital. He was then partially paralysed on the right side, with anæsthesia and analgesia of the same side; and partial loss of the special senses, the sight being imperfect in the left eye. He gradually improved, and at the end of three weeks was able to walk about. He said he felt much better after the application of silver coins to his left side. He then heard of the sudden death of his father. He was seized with pain in the head, sickness, and return of all the symptoms which he had on admission. He again gradually recovered, and when he was able to walk about left the

hospital. A short time afterwards a fellow-servant called on me to say that the young man had had another attack accompanied by all the old symptoms.

The following case I have already related in these 'Reports,' but again quote it as showing the hysterical nature of the complaint, as it would have been called, had it occurred in a woman :

CASE 7.—H. A. T—, æt. 22, a Swiss, employed in an equestrian circus. He had often had falls, and received various contusions on the body and head. He had been out of his occupation for a fortnight, wandering about the streets and having scarcely anything to eat. On the evening of admission, when in the street near the hospital, he felt a rush of cold in his right arm, he then shook violently and fell down. He was picked up by the bystanders and brought in within a quarter of an hour after the occurrence. He was pale and motionless and thought to be dead. He was found, however, to be unconscious only, and was put to bed, when within an hour his senses returned; he then related the circumstances of his attack, and was observed to be in the same condition as I found him a few hours afterwards. He was quite sensible; the right arm was tightly flexed across the chest, the elbow- and wrist-joints being bent, and the muscles rigid; the hand was turned outwards, the fingers separated, the thumb drawn in, and the little finger tightly flexed, whilst the last phalanges of the other fingers were extended; it thus appeared as if the muscles supplied by the ulnar nerve were especially in action. The foot was extended and the muscles contracted; he could stand on the limb, but moved it as if it were a wooden one. There was no apparent paralysis of the face, but he said he could not whistle. Besides this spasmodic contraction of the right limbs he had almost complete anæsthesia of the same side; he could not feel when touched, nor discern hot from cold. This loss of feeling included the whole of the right side of the face with the eye and nose and the trunk as well as the extremities; he had lost also the senses of smell, taste, and hearing on that side. He said he had a pain in the right temple and forehead. On testing with

both forms of galvanism it was found that the muscles did not react so well as on the healthy side.

At the end of a week the sensation was returning in the face and body, but was still very imperfect in the limbs where the rigidity still remained; hearing and taste had returned. He still gradually improved, and at the end of another week he could extend all his fingers but the ring finger, and the little finger still remained flexed. The leg was weak though he could walk with it. Sensation was still impaired. About this time he was said to have had a fit in the night, when it was observed by the house-physician that the whole of his original symptoms had returned, including the rigidity and anæsthesia, but on the following day he was as well as before the attack. Two days after he left the hospital surreptitiously with some of his neighbour's clothes, thus confirming the suspicion formed from the young man's own history and general demeanour that he was thoroughly depraved and vicious.

I will now give an epitome of two cases of hemianæsthesia, reported in full in the '*Revue de Médecine*' and in '*Neurologie*' (1882) respectively. It will be seen that according to the modern theory both were diagnosed as cases of apoplectic effusion. In one no disease was found after death, and in the other recovery took place in a few hours.

CASE 8.—Th., æt. 45, journalier, entered La Charité March 27th, 1879, under M. Vulpian. A fortnight before, while at work, he fell down with loss of consciousness. How long he remained in this condition he did not know; when he got up he felt giddy, and dragged his left foot in walking, and his left arm was more feeble than his right, and he could not see well with the left eye. He spoke as usual and understood everything that was said. When he arrived home he complained of a pain in his chest and a feeling of stifling which was relieved by a bandage tied round his chest.

On entering the hospital he walked more feebly with the left leg, and his intellect did not seem very bright; his left arm was weaker than the right. The skin of the left side, including the body and limbs, was totally insensible to touch,

pinching, and faradisation. Taste was abolished; salt, sugar and quinine were not appreciated. Hearing was intact. The sight of the left eye was much confused; the palate insensible. He had irregular choreic movements in the left limbs.

Faradisation was ordered and in a few days he began to improve. On April 6th sensation was returning in several parts, and the eyesight was improving. Strychnia and iodide of potassium were ordered as well as galvanism. On the 28th sensation was returning in all parts, and on May 11th the patient left the hospital considering himself well.

M. Vulpian commented on the case, and gave it as his diagnosis that it was a case of cerebral hæmorrhage, and from a consideration of the symptoms and the number of parts involved that the seat of the hæmorrhage was in the cerebral peduncle or posterior part of the internal capsule. He attributed the recovery to the absorption of blood, and to the new routes found for the passage of impressions.

The patient, after leaving the hospital, became more dissipated in his habits, and was continually inebriated. He died rather suddenly in November, 1880. At the autopsy the brain was examined with the greatest care and no lesion whatever could be found. "On n'a pas trouvé le moindre foyer d'hémorrhagie ou de ramollissement, soit dans l'écorce grise du cerveau, soit dans les corps opto-striés ou dans les capsules internes, soit dans les pédoncles cérébraux."

CASE 9.—C. J—, a tailor, was struck down by an attack of apoplexy and taken to the Hôtel Dieu. The case is reported by Dr. Debove.

When he had recovered his consciousness it was found that he had lost the power of his left side as well as sensation. He was just able to walk, but he could scarcely move his arm. The face and tongue were also slightly drawn. Sensibility was totally abolished, both to local pain as well as to heat and cold. Taste was also impaired on the left side as well as smell, and hearing was also affected. The sight, too, was impaired as regards colours, all these appearing as black or grey. The diagnosis was cerebral hæmorrhage.

Six days after the attack of apoplexy two magnets were applied, one to the middle of the chest and the other on a

level with the knee. The patient soon afterwards felt a violent pain in the head, and in two hours' time sensation was returning. In another half hour sensation had returned, as well as the functions of all the special senses. In the author's own words :

"Mars 22 (6 heures du soir).—Six jours et demi après l'attaque d'apoplexie, nous constatons de nouveau la persistance des troubles moteurs et sensitifs, la sensibilité générale est toujours abolie du côté gauche, les sensibilités spéciales sont toujours aussi profondément atteintes. Puis après ces constatations nous appliquons au côté gauche deux aimants l'un au niveau du thorax, l'autre au niveau du genou.

"A 6½ le malade ressent, dans le côté gauche de la tête principalement, une violente douleur.

"A 7 heures la cephalalgie s'est dissipée.

"A 8 heures la sensibilité et la motilité commencent à renaître.

"A 8½ les aimants sont enlevés et nous procédons de nouveau à l'examen du malade.

"La sensibilité générale est revenue dans tout le domain précédemment anæsthésié. Les objets sont nettement distingués, les couleurs nettement reconnues, les sensibilités gustative, olfactive, auditive, sont également intactes et complètes.

"Tout trouble de la motilité a disparu. Le malade marche et même court facilement sans traîner la jambe."

The next case is one of interest as showing the functional nature of hemianæsthesia and its association with much moral obliquity. The difficulty of distinguishing genuine symptoms from feigned is always excessively great in hysteria, but there is less object in endeavouring to make the diagnosis if, according to my theory, the physical want of feeling, want of power, or blindness, have the same foundation as the mental dulness, loss of will, or moral blindness. The supposition of imposture will not account for the symptoms which were present in this case, nor should such a view be maintained because of his miraculous cure, since almost as speedy a cure was effected by means of magnets in the case last related.

CASE 10.—Albert R—, æt. 36, admitted under Dr. Wilks, July 16th, 1881. The case reported by Mr. F. Eastes. He

was brought into the hospital by a policeman who picked him up in the streets insensible and occasionally convulsed. He afterwards gave the following account of himself:—He was a Frenchman, by birth, and had been in the army. Ten years ago he went out to Australia and been employed in a botanical expedition by the Victoria Government. He was a widower and had one child. He had a brother who suffered from tetanus after a bullet wound, but who recovered; he saw him in his tetanic paroxysms. He had lived long amongst the natives of New Guinea and had several wounds in fights; he had also killed several. He did not drink but was fond of smoking. He sailed from Melbourne for the purpose of visiting his daughter in Paris, when on June 10th, just before reaching Plymouth, he fell down the cabin steps and immediately vomited blood; his abdomen afterwards became black and painful. He was attended to by the doctor, and when he arrived in London was sent to the London Hospital. Whilst there he vomited blood and passed the same by the bowels and with the urine; soon afterwards he fell down and was paralysed. He was then transferred to a medical ward. He had morphia injections and gradually improved, so that he walked out of the hospital on July 13th. On the following day he was walking to London Bridge to start for Paris, when he fell down and was brought to Guy's. The above account is his own, but from what was subsequently seen of the patient, it cannot be relied upon. The house-physician of the London Hospital, Mr. Neatley, was written to about the case, and he replied that the patient was admitted with the symptoms he described, but soon made the most complaint of his head where he said he had excruciating pain at the site of an old wound. Hypodermic injections were continually used to relieve him. He occasionally had fits and loss of power in his limbs with speedy recovery; the ophthalmoscope showed nothing to account for his occasional unilateral blindness. Dr. Hughlings Jackson did not express any opinion about the case.

On admission into Guy's he was seen to be a strongly built man with coarse black hair. He had a small scar over the left parietal bone, and here he experienced great pain on pressure.

The left arm and leg were paralysed but the face was

unaffected, and he could protrude the tongue to either side. He had complete anæsthesia and analgesia of the whole of the left side, including the trunk, face, nose, lips, tongue, &c. With the left eye he could only distinguish between light and dark, and could not perceive colours. He could not hear a watch tick when pressing against the left side of the head; could not perceive with the left nostril oil of cinnamon or liquor ammoniæ; could not taste on the left side quinine, sugar, tartaric acid, &c. The senses on the right side were natural. His spine was very sensitive. Many of the reflex actions were wanting on the left side.

In the evening he had a convulsive fit; he breathed stertorously and threw his right arm and leg about, the left limbs remaining quiet; the tongue lay coiled up in his mouth so that it had to be drawn forward to prevent his choking; his pupils were dilated. On becoming conscious he cried out with pain. The urine had to be drawn off. He had an injection of one sixth of a grain of morphia and went to sleep, but woke after three hours, and had another convulsive fit. During the following day he had several choking fits and his tongue became curled up, requiring force to draw it forwards. He was injected several times with morphia and slept. At intervals he would wake and cry out with pain in his head.

On July 19th he was so noisy that he was transferred to the strong room. He remained in much the same state for two days, constantly calling out and having morphia injected. Although it was at first thought that he might be suffering from some local disease of the skull, membranes, and brain in consequence of the injury, his strange manner raised a suspicion of malingering. On July 21st water was injected into the arm instead of morphia, when he became immediately quiet and remained so for some hours, talking sensibly and asking to be sent back to his ward. The anæsthesia remained in the left side, nor could he feel a test-tube filled with boiling water applied to the skin on the left side of the body. During the next few days he had the subcutaneous injection of water constantly used, and metals were applied to his arm and leg. He was soon able to move his limbs a little, and on July 27th was sitting up in a wheelchair. On July 30th Dr. Steele informed him that he had received a letter from the Duchess

of Sutherland inclosing a sovereign and saying that she had some money for the patient and wished him to come and fetch it. In the evening he complained of great pain in the left leg, and when it was examined he was found to move it almost as well as the other. On the 31st he got up and walked up and down the ward by the aid of another patient, but said he could not move the arm, the limb falling when it was raised. He was, however, seen to use it occasionally to support himself. He was then told he could not leave the hospital on that day. In the evening he could scarcely walk.

August 1st.—Being allowed to go out to see the Duchess, he got up and walked about. During the next few days it was found that sensation was slightly returning but the special senses were still dull. He left in the middle of the month; he was able to walk very well and to move the left arm partially, but the anæsthesia had not quite departed. He said he was well enough to work and left with that object.

I heard no more of this patient until my attention was directed to his name in a French religious journal of October 7th, 1882, called the '*Rosier de Marie.*' In this there was an extract from another paper styled the '*Journal de Lourdes,*' which gave an account of a pilgrimage taken to Lourdes by the Archbishop of Cambrai and numerous followers. Amongst these was our patient, who had been an inmate of the hospital at Lille. It will be seen that all his symptoms had returned and that he was obliged to have recourse to a wooden leg as well as crutches. The statement is not correct that he was shown to the International Congress, since the only member of that body who saw him was Dr. Lancereaux when he visited Guy's with me. It will be seen that after descending into the water at Lourdes, the archbishop and the people looking on, he dropped his wooden leg and crutches, and walked out of the grotto cured.

I extract from the paper the account of what happened after the patient had descended to the spring, and the words of the archbishop:—" '*Mes frères,*' dit-il, *les larmes aux yeux, 'récitons deux dizaines de chapelet pour nos malades du pèlerinage de Cambrai.' Comme il finissait, un cri partit des piscines et bientôt après un homme jeune encore sortait chancelant d'émotion, et portant dans ses mains deux béquilles désormais inutiles.*

“C’était Albert Rose de l’hôpital de Lille atteint d’hémiplégie et d’hémianæsthésie du côté gauche à la suite d’une opération du trépan mal réussie; il ne marchait qu’avec deux béquilles et le genou gauche appuyé sur une demi-jambe de bois. Il avait aussi perdu l’usage de l’œil gauche. Les médecins du congrès médical de Londres l’avaient déclaré incurable. Plongé dans la piscine pendant que la foule priait au dehors avec le saint archevêque puis tout à coup sa jambe s’étendit et son œil gauche s’ouvrit à la lumière. Il était complètement guéri.”

On reading this miraculous cure I wrote to Dr. Bèchamp, of Lille, who was good enough to give me all the information he could gain from the hospital doctors. They were reluctant at that time to give their opinions about the case, as it was under investigation. It appeared, however, according to an extract from one of their letters, that when the man went back to Lille all his symptoms returned and that he went into another hospital.

“Il a été traité dans mon service pour des accès épileptiformes et une hémiplégie avec hémianæsthésie. À sa sortie son état n’était pas notablement amélioré. Il se rendit à Lourdes et fut subitement guéri en présence de plusieurs témoins. A son retour à Lille le mouvement et la sensibilité étaient revenus. Je ne le constatai pas moi-même puisque j’étais absent, mais plusieurs de mes élèves qui le connaissaient le constatèrent. On l’a perdu de vue pendant plusieurs semaines et avant-hier une lettre m’apprenait qu’il était entré à l’hôpital de V., présentant les mêmes accidents qu’à Lille et qu’il cherchait à tromper sur son identité, prétendant que le miraculé de Lourdes est son frère et non point lui.”¹

¹ In the ‘Contemporary Review’ for Nov., 1882, is a paper by Mr. F. Clarke (S. J.) on “Modern Miracles.” He refers to the cures taking place at the fountain at Lourdes, which, as is known, has received the sanction of the Bishop of the diocese. He describes many, and amongst others the following, which is worthy of note by the believers in metallotherapy. A woman from Louvain had been paralysed on the left side for seventeen months, her leg dragged helplessly along the ground, she could not move the left arm, and she could scarcely see or hear with the left eye and ear (there is no mention of smell or taste, probably these were not tested). “Whilst praying fervently in front of the grotto all at once she cried out, ‘Sister Pauline, my fingers are moving.’ A few minutes after a sharp pain pierced her arm and side, and she feels that she is cured. The

To illustrate the true *rationale* of cure in these cases, I will give one more example in which "neglect" effected in a month what systematic treatment could not do in a year.

CASE 11.—E. M—, a female school-teacher. For two or three years she had been ailing with a number of nervous symptoms. She had often lost the use of her limbs, or lost feeling in them. She had had headache, sickness, pain in the back, scanty and irregular menstruation, and also "mad fits" or "screaming fits." On admission she said she could not walk, and it was found that she was quite hemianæsthetic and hemianalgesic on the right side. She could not feel her needle when she grasped it, nor did she show any evidence of pain when it was thrust into her arm. This want of feeling included half of the body as well as the mucous membranes and conjunctiva. The senses of sight, hearing, taste, and smell were also imperfect. Two sovereigns were tied round the leg without effect. Next day four sovereigns, previously wetted with salt and water, were laid flat on the arm in a circle, three quarters of an inch from one another and tied on with a piece of tape. She was watched carefully and tested repeatedly for some hours with no result, being perfectly hemianæsthetic and hemianalgesic the whole time, whilst the corresponding part of the opposite arm retained its normal sensation to pain. In the same manner silver, copper, zinc, tin, lead, and iron, and various combinations of these were respectively tried day by day without any result. She remained in the hospital seven months, using galvanism and medicine of all kinds, when she was discharged in the same state as on admission.

Some weeks afterwards her mother, who had to keep her daughter, requested me to take her in again. I did so, and then determined to follow my own and well-tried method—that was to give her some moral discipline and leave all medical treatment alone, as it so often perpetuates hysterical ailments. I ordered her nothing and systematically passed by her bed saying in her hearing that I could give no more trouble to her case whilst so many persons really ill required my attention. same afternoon she appeared before the Commission appointed to examine alleged miracles, and in the presence of two physicians, walks, runs, carries heavy objects about with her left hand, sees perfectly with her left eye, and hears perfectly well with her left ear."

In fact, I neglected her for a purpose, when one day, after two or three weeks' time, I found her out of bed and sitting dressed in a chair at its side. I then spoke to her, and she told me that she could walk a little, and she thought she was regaining some feeling in the right side. I expressed my satisfaction at the turn her case had taken, and hoped she would now speedily get quite well. This she did not fail to do, as she daily grew stronger, and soon left the hospital quite well.

The case exemplifies what every medical man must have seen, not only that in hysteria and some nerve disorders medicine is useless, but that it often does positive harm. Whilst this girl was under treatment she made no improvement, and when the case became interesting on the introduction of the new system of "metallothérapie" all chance for her was gone. "Neglect" was the treatment she needed. It was more than she could withstand. Her reflections on finding herself in bed for a fortnight with no one caring for her roused her dormant will, and was therefore exactly the stimulus she required.

In some of the cases which I have related, there has been a functional hemiplegia accompanying the anæsthesia, whilst in others the apparent weakness has been due only to the want of co-ordination resulting from the absence of feeling. In all these cases, however, where the hemianæsthesia has predominated and been more or less complete, the disease has been of a functional character; this is also true as regards all the cases which I have read as recorded by others. It has sometimes come on insidiously in nervous hysterical women and at other times has had its origin in a physical or moral shock. From whatever cause arising, my explanation of the nature of the disease is that one hemisphere has become more or less functionally inert. It will be seen from a perusal of my cases that the remedies have been of various kinds, the most striking those which have suddenly brought the brain back again into working order. The remedies have been ordinary medicines of every description, the continued application of metals, of galvanism, of magnets, and solenoids, the application of discs of wood and mustard plasters; also the use of the diapason, the waters of Lourdes, charms,¹ and simple

¹ In considering the action of medicines in affections of the nervous system we must not ignore the great influence which some remedies exert on the mind.

neglect. It is only therefore reasonable to consider the relation between the malady and all these modes of cure before endeavouring to frame a theory as to their *modus operandi*. I will recommend this consideration to those of the French School who have formed theories on the nature of the cure of hemianæsthesia by the study of metallothérapie only. As an example of the latter, I will refer to the case of Albert Rose where a sovereign retained in the pocket had a more striking effect than the temporary application of gold to the skin or its administration in minute doses internally.

My own opinion I have already expressed as to the arrest of the function of the brain and its sudden starting again into action. In my former paper I illustrated this by the story of a watch. This was one of the earliest I remember, it was in the possession of a school-fellow, and the envy of all the other boys. It had this remarkable property, that when the owner took it from his pocket, although it might give the right time, it was found to have stopped. The sudden pulling it from his pocket had arrested its movements. He would then give it a sharp knock and send it on again. The balance was in a state of unstable equilibrium, and was thus ready to stop or move on again under any jarring influence. The brains of these poor hysterical people seem in like manner to stop working on receiving a shock whilst another shock will start them again.

Thus, in the same review which contains Mr. Clarke's paper on "Modern Miracles," there is another by Dr. Jessopp on the "Superstitions of Arcady." Amongst other stories he relates how, on visiting a cottage and seeing a charm round a dead woman's neck, he obtained the following explanation from the husband: "You see, sir, as my wife she wore allus an ailin' woman an' doctors' stuff did her no manner o' good, and she'd giv' it over an' a year or two ago she says to me, 'Joe,' she says, 'You mun' go to a cunnin' man for me, him at Shawby as they du talk as surprisin.' So I took a day an' I went and I found him out and I told him all about my old missus and he never said a word till I'd done talkin', and then he didn't say much. But, says he, as though he know'd all about her, 'Oh, ah,' says he, 'She's got the gripes occasional and a sort of numbness like! No doctor's stuff won't touch that,' says he, and turned away and he sat down and lawk and behind him there was a heap of grit books, and he put on his glasses and he began to turn 'em over. I 'aint no scholar myself, but bless ye, I could easy see they warn't like other books. And then he wrote the first o' them charms. He never giv' her no medicine, all the time as ever I went to him, only one of these charms, and its surprising the deal o' good they done her, though you mayn't credit it. They always seem'd to revoive her like!"

SATURNINE LUNACY.

BY JAMES F. GOODHART, M.D.

CASE 1. *Lead poisoning; mania.*—Elizabeth W—, æt. 29, living at Camberwell, was admitted into Guy's Hospital, under my care, on February 15th, 1879. Her father had had rheumatic fever, and her mother and one sister died of phthisis. She had always been delicate, and when very young she had enlarged glands in her neck which discharged. Seven years before her admission she had rheumatic fever. She had been married six years, and had had four children and one miscarriage, only one child being now alive. She had never had any delusions, hallucinations, or cerebral symptoms of any kind during or after her confinements. Two years before her admission she had some eruption on her skin which a visit to Margate quite cured.

The illness for which she was admitted began seven weeks before with severe pains in her stomach. This came on quite suddenly and had been gradually getting worse. She was thought to have hysteria by her doctor, she had occasionally vomited, and her bowels had been confined.

When she came into the hospital she was very anæmic. She complained of pain whenever and wherever she was touched, and she spoke only in a hollow whisper. She was rather deaf, complained of buzzing in her head, and talked a great deal of rubbish; but there was no paralysis, and from the fact that she had old scars about her legs and that her skin felt nodular, I anticipated the

possibility of some syphilitic meningitis, which is often chronic, as her ailment appeared to be. It was subsequently noticed that her wrists were weak and her shoulder muscles also, but her chief troubles were that she had much pain in all her bones, and she had attacks of delusions which she called "dying fits," in which she imagined that certain parts of her body were dead. She was treated with iodide of potassium and she gradually improved. The existence of lead-poisoning was not at first suspected as there was a want of definite affection of any group of muscles, and the vagaries of the mind attracted much attention.* However, it subsequently became evident to Dr. Moxon, into whose hands she passed, that the extensors of her wrists were unduly weak, and he found a distinct blue line upon her gums. Search was now made for a cause of lead-poisoning, and it was elicited from her husband that for a considerable period she had been in the habit of using acetate of lead lotion as an application to an eruption on her skin. The iodide of potassium was increased to twenty grains three times a day, but with no further effect. She passed into a condition of religious melancholia, became very troublesome, passing her evacuations in the bed, and sweating profusely. •She would occasionally have lucid intervals and then relapse again, trying to escape from the ward, or sitting down in an abject state, talking all sorts of nonsense, born of religious mania. And so she continued till she left the hospital, to all appearance a confirmed lunatic, on July 26th, after five and a half months' treatment.

I have attempted to trace her since with the object of giving her subsequent history, but have been unable to find her, as her husband does not now reside at the address given in the hospital book.

CASE 2. *Paralytic dementia in a painter.*—Henry C—, æt 28, was admitted into John Ward, under my care, in 1880. His father is a painter, has a blue line on his gums, but has never had any symptoms of lead-poisoning so far as can be elicited from him. The patient is also a painter but is not known to have had any symptoms of poisoning. Three years ago he fell down a ship's hold and struck the back of his head, but this is not known to have permanently injured him in any way as he was perfectly well afterwards. He is a sober man

and has always enjoyed good health till a year ago. He then became desponding and was obliged to give up work. He next became the subject of fits of unconsciousness, lasting for several hours ; and for hours and sometimes days he would lie with his left side paralysed. In the few months before admission he had led a purely animal existence, taking no interest in his surroundings, but having a good appetite.

He lay in bed in a stupid condition ; he would occasionally after some time pull his wits together to answer some simple question, and could tell his name and age ; the day, the month, and the year, were quite beyond him.

His mouth and tongue twitched. He had a fair muscular development but his grasp was feeble indeed. His hand was very tremulous when he attempted to write. His sight was good and his optic discs were natural, the pupils being moderately and equally contracted. His viscera were all healthy.

Now this man had not at the time of his admission any symptoms of lead-poisoning, but then he had done no work for twelve months, so this was hardly to be expected. But one could not help suggesting that there was some connection between his work and his disease, and my colleague Dr. Savage, who saw him, accepted this view as probable.

He was removed from the hospital after a short stay and when we thought he was improving, because he was troublesome ; and I heard afterwards that he became the inmate of a lunatic asylum.

CASE 3.—William H—, æt. 38, was admitted into Guy's Hospital, under my care, on January 6th, 1882, for violent epileptiform convulsions.

His history is a very interesting one. He is a metallic cast maker, working principally with sheet lead. His father who worked at the same occupation, died at 60 of paralysis, but there is no history of gout.

He has enjoyed good health till within the last few years. Since then he has had frequent attacks of lead-poisoning. He was under my care in Guy's, in 1878, for violent epileptiform convulsions, and for a maniacal state supervening, and rapidly got well under treatment by iodide of potassium.

He was again in hospital in the winter of 1878, and again in

1879. In 1881 he was re-admitted for lead colic. At this time, under my colleague Dr. Fagge, his nervous system is described as "mental faculties clouded, speech thick, no abnormalities of sensation or motion." While under treatment he had half a dozen epileptiform fits, four of these occurring on one day. The fits were followed by unconsciousness and stertorous breathing. In these attacks he slowly regained consciousness; appearing stupid for some time after. But he left the hospital after a three weeks' stay quite well. He now went on without illness till the end of the year when he began again to feel colic, and this got so bad that he had to leave off work. On January 1st he had two fits, and when another occurred on the 6th his friends brought him at once to the hospital. He was very pale, somewhat thin, had a well-marked blue line upon his gums, and was very drowsy but answered questions slowly and correctly when roused. He had another fit in the afternoon, which commenced with a peculiar low cry, quickly followed by a tonic spasm producing rigidity of the body, which was in turn quickly succeeded by clonic spasms of all the extremities. When the fit was over there was coma with stertorous breathing, and this curious feature that the right ala nasi was drawn tightly against the septum in inspiration. Ere he had well recovered from the first a second fit ensued with similar phenomena; in each a nitrite of amyl capsule was administered with the object of dilating his cerebral vessels and thus arresting the convulsions. The volume of the pulse increased but no other appreciable effect was produced. After the fit he was very violent and it became necessary to remove him from the ward. He was constantly and actively delirious with all sorts of delusions, and for some days he had a full soft pulse and a profusely sweating skin. In fact the case was one to all intents and purposes of delirium tremens, and I had to re-discuss my original diagnosis of lead-poisoning, convulsions, and subsequent mania, and felt bound to acknowledge the possibility of the existence of alcoholism. Accordingly the treatment of iodide and bromide of potassium, to which he had previously been subjected was discarded, and opium, digitalis, and strong and copious liquid nourishment were ordered in their place. Food he at all times took ravenously, till at one time he was taking five pints of milk as well as three eggs and beef tea.

But he got worse notwithstanding. He passed his evacuations under him, his pulse became rapid and weak, and I now ordered him brandy \mathfrak{zss} every two hours. Still, without any obvious improvement, his delirium, restlessness, and delusions continued all the same. Ten days after admission his mind cleared a little; he allowed the nurse to attend to him without struggling, and when spoken to he seemed to understand although he could not find words to express himself. He would endeavour to find an answer and then laugh at his inability. From this time he had occasional lucid intervals, his general condition being a quiet muttering delirium from which when spoken to he would rouse himself and listen, and occasionally answer questions rationally, and then again relapse into a noisy, maniacal condition very difficult to manage. One of his hallucinations was that water kept dropping on to his head; another that we were in league to murder him; and a third, and rather persistent one, that we kept a coffin near his bed. He thought this particularly unkind on our part, and assured me that this alone kept him from getting well.

It will be unnecessary to prolong the details, suffice it to say that, when at the end of a month, he remained in much the same condition as when he was admitted. It was hardly possible to allow any longer that his case was one of delirium tremens, and I decided to go back to the treatment by iodide of potassium, and this was subsequently persevered in. Even then he remained in the same condition till the end of the sixth week, and I accordingly asked my colleague Dr. Savage, of Bethlem, to see him. He thought the view I had taken of the case in calling it one of saturnine lunacy was the right one, the speech and the hallucinations being like other cases he had seen of that kind.

Soon after this (on February 18th) his mind cleared rather suddenly, all his hallucinations vanished, and he said he thought his head was getting all right again, and so it did. He left the hospital quite well after about two months' stay.

But it is important to note that he has now been in the hospital five times, that he has had severe convulsions in all his attacks, and that the last state of the patient is far worse than the first; and no one can doubt, who studies his history attentively, that if he continues to follow his employment he runs a great chance of becoming a confirmed lunatic.

CASE 4.—*Lead poisoning; maniacal delirium; delirium tremens; pneumonia.*—Cornelius D—, æt. 30, lives at Rotherhithe; he was admitted on March 6th, 1880. Has been a hard drinker. Has had pains in his abdomen for many months, also cramp in his arms and soreness of gums. There was a well-marked blue line on his gums, and he had some pneumonia at the left base, though his temperature was only 101° , pulse 102, resp. 30. The night after his admission he became violently maniacal and had to be removed from the ward. He was quieted by the administration of hyoscyamine, but his condition now resembled one of delirium tremens. His pneumonia gradually cleared up, and he left the hospital much improved, after six weeks' stay. He was at first treated for his pneumonia and subsequently with iodide of potassium and iodide of iron.

In this case it is doubtful how far the maniacal delirium was due to the pneumonia and how far to the lead; but as maniacal delirium is one form in which lead-poisoning clothes itself, and the man was suffering from lead, I put it with the other cases.

As there are not many of these cases on record, a few remarks upon their points of interest will not be out of place. They will mostly centre round the symptoms, but I may perhaps say *in limine*, as regards the relation between lunacy and lead-poisoning, that these and other similar cases are sufficient to prove some connection between the two; sufficient perhaps to prove more than this, that insanity of several forms is the direct result of chronic lead-poisoning in some people. Now, I think that fact is worth noting, because it throws light upon the oft disputed question whether dyspomania, for example, is a primary nervous fault or the result of excessive indulgence. I suppose I am not wrong in stating that the general tendency nowadays is to consider the nervous system primarily at fault in all lunatics, and exciting causes as hardly competent alone to work such mischief. This is, of course, a very intricate question, as any one can see who takes the trouble to reflect, not upon the laws of thought in the abstract, but upon the *habits* of thought as they occur in his own mind. Surely a man can educate himself into some forms of lunacy as truly as he can by careful training make for himself a continually pro-

gressing intelligence. If so, insanity may be induced without any predisposing causes of any kind, and the production of insanity by alcohol and lead I take to be confirmatory of this line of argument.

To my mind this is a position which is full of solace. There is in the public mind an exaggerated horror of insanity, such as makes it regarded as a blot upon the family escutcheon. Your own brother may drink hard all day long, or be perpetually floating on the cracked bubble called credit, and all that we say of him is "Poor old So-and-So, a very good fellow, but too fond of his beer;" or, "What a pity it is A or B is always under water"; but, be he a lunatic the sooner out of sight and mind the better, and he becomes a subject of conversation as much tabooed as if he were an inmate of one of Her Majesty's prisons.

Insanity is truly repulsive for what it is and what it does in spoiling the fair face of nature, but it need not be made worse than it is; and once let it be recognised that when not the result of actual disease it is in some measure the outcome—if not of a man's own choice, at least of his self-neglected mental education—surely the number of lunatics might be materially diminished, and a load of anxiety would often be lifted from the shoulders of the relatives of the insane.

But now, as regards the symptoms of these cases, it appears not only from them but from others which have been recorded by Drs. Rayner, Savage, Barton, Hammond and others, that they are remarkable in the frequency with which they resemble those of general paralysis of the insane or the impairment of intellect with feebleness of gait which is characteristic of acute or chronic alcoholism. The resemblance to general paralysis is seen in the thickness and hesitancy of speech, and in the loss of memory, and perhaps in the paralysis; but it is not close, as all the more characteristic symptoms are absent. The resemblance to delirium tremens, however, has been in several cases most striking, and as I have mentioned in my third case particularly it was impossible for some time to decide positively, this resemblance was so exact. Dr. Savage mentions another case of the same kind. A man was admitted for what was supposed to be delirium tremens, and it turned out that he was a most sober man, but that he was suffering from lead-poisoning.

Acute mania is not uncommon, it was present in two of these

four cases, and it may terminate in a condition more or less resembling dementia such as existed in three out of these four cases.

This resemblance between the cerebral symptoms of lead-poisoning, of alcoholism, and of general paralysis, is well worth remembering not only for purposes of diagnosis, but for treatment, and also for rightly estimating the pathology of the first.

For treatment it is all important, as a prolonged course of iodide of potassium, usually held to be one of the best means of eradicating the taint of lead, could hardly be considered suitable for delirium tremens where food is even more necessary than drugs, the administration of opium perhaps excepted.

But the resemblance is most suggestive of all when we come to consider the pathology of lead lunacy. We know of general paralysis of the insane that it is associated with chronic meningitis of the cerebral hemispheres, particularly over the frontal and parietal convolutions, so that when the membranes are stripped from the surface of the brain those parts remain scarred and dimpled from the wasting of the cortex, or its adhesions to the vessels of the membranes. We know of chronic alcoholism that it produces wasting of the cerebral cortex, so that the membranes become opaque and white, and the convolutions shrink away from each other, leaving widely patent sulci.

On the other hand, we know of lead, first from experiments on the lower animals, that it produces fibroid changes round the vessels; and in this way, as has been demonstrated by Charcot in guinea-pigs, can produce a granular kidney: secondly, we know as a clinical fact or pathological sequence, that chronic lead-poisoning is often associated with granular kidney.

It would seem, therefore, putting altogether aside the clinical features of the three, that a study of the pathological changes known to occur in each, affords strong *primâ facie* evidence that the changes in the brain are not very dissimilar in all, and a presumption therefore in favour of the symptoms being more or less alike in all.

But I must push this argument one step further for the sake of the treatment of these cases. Of the pathology of general paralysis I can say no more, for but little more is known. But

the resemblance between the working of lead and alcohol is strikingly close. Both poisons are associated with vascular tension, both are associated with gout; both are associated, oftentimes with violent epileptiform convulsions and with chronic dementia, and both are associated with granular kidney. But while we know with a fair amount of certainty that alcohol persistently adulterating the juices leads to arterial spasm, and later to persistent contraction and thickening of the vessels, we are not so sure about lead, and some believe that its action is a special one upon the nerve-centres. I would, however, insist that the results of lead-poisoning so much and in so many points resemble alcohol poisoning that in all probability their action is the same; and in the epileptiform convulsions or the *acute* mania of either we see the nervous discharges of the anæmic convolutions of *acute* arterial spasm, in the more chronic delirium dementia or paralysis the impaired nutrition of a wasted cortex due to the crippled circulation of permanently thickened and diseased vessels.

This point has a direct bearing upon the treatment of such cases. If they be due to arterial spasm, obviously relaxers of spasm and aids to the circulation should be the remedies to help us. Therefore opium, digitalis, perhaps even alcohol itself, in appropriate cases, may be expected to bring relief, and to these may be added, without by any means exhausting the list, nitrite of amyl and nitro-glycerine; drugs which have been brought forward in recent times, and have certainly had considerable success.

As regards the exhibition of iodide of potassium to procure the solution of the lead deposited in the tissues and its discharge from the system by way of the urine, it is worth remarking that although such is, I believe, the very general practice, and lead under its administration has certainly been detected in the urine many times, the statement has been made by Dr. McDowell that at Newcastle, where many bad cases of dementia from lead-poisoning occur, it has been found that the iodide treatment poisons the patient outright, coma and rapid death supervening. Dr. Hilton Fagge has published a case in the 'Transactions' of the Medical and Chirurgical Society, in which a lead line appeared on the gums after the administration of iodide of potassium, and a case pointing in a similar direction

has quite recently been recorded by Mr. H. Morris,¹ at the same society, where a patient with lead in her system, but without any lead line or other symptom of plumbism, developed both lead colic and a blue line on the gums during a course of iodide of potassium.

In conclusion, let me say that although the somewhat rare cases I have narrated have an intrinsic interest which makes them worth attention, they have a much wider value if they serve to remind us that what stands out here as a cause of unmistakable disease, may have, probably has, considering the many uses to which lead is put, more subtle workings—far more obscure symptoms—than those to which our attention is compelled by their severity and upon which they may serve to stimulate inquiry.

¹ 'A Case of Aneurism of the Arch of the Aorta involving the Innominate Artery, with Remarks on the Distal Ligature.' Read at the Meeting of February 13th, 1883.

ON

ACUTE GONORRHOÆAL RHEUMATISM.

BY J. N. C. DAVIES-COLLEY, M.C.

RATHER more than four years ago I published in the 'Obstetrical Journal'¹ a series of cases of rheumatoid inflammation of the joints. The subjects of the disease were all women below the middle age, and the exciting cause in each case appeared to be some vaginal or uterine irritation. Three of the cases were pregnant. I was therefore led to suppose that the disease was peculiar to women, and that it was different from, though probably allied to, gonorrhœal rheumatism. Subsequent experience has caused me to modify both these opinions. At least three cases of a similar affection in the male have come under my care since I wrote the article; and my observation of the symptoms found in these and other cases has induced me to regard the affection as a form of gonorrhœal rheumatism, the peculiarities of which have been omitted or only slightly alluded to by our best known surgical writers.

At the same time I would use the term in so wide a sense that it should not be understood that the discharges from the genital organs which give rise to these rheumatoid inflammations are necessarily gonorrhœal. In one of the cases which I have to relate there appear to be some grounds for the belief

¹ June, 1878.

that the disease was set up by a balanitis, and not by an ordinary gonorrhœa; and in women I have sometimes thought, though I have found it difficult to prove, that the determining cause has been of a leucorrhœal character.

The usual description of gonorrhœal rheumatism is that it affects especially the knee-joint, rarely attacks women, and that it resembles the chronic forms of synovitis. The patients are said to be generally weak and anæmic. They often suffer at the same time from tenderness of the soles, and some inflammatory affection of the iris or sclerotic. The pain is not very acute, but rather of a dull aching character, which becomes worse at night. The disease is tedious, but, as a rule, it disappears with the gleet, by which it has been set up, and the joint then completely recovers.

Thus, Prof. Billroth¹ states that "it almost invariably attacks the knee," that it "generally occurs upon both sides," and that it is a "subacute serous synovitis, which generally terminates in complete recovery of the joint provided that the patient will keep at rest and avoid all further irritation of the urethra."

Mr. Holmes² says that "it almost always affects the knee; other joints may also be implicated, but I (Mr. Holmes) can hardly recollect a case in which the knee was not the principal seat of disease." "It is far rarer in women than men."

According to Mr. Bryant's 'Surgery,'³ "at the end of an attack of clap patients without doubt are often attacked with severe pains and tenderness of one or more joints, attended with effusion and constitutional disturbance." . . . "The knees and ankles are the joints mostly involved, yet those of the upper extremities are so at times." A somewhat fuller, but on the whole similar account, is given by Mr. Bond in a lecture published in the 'Lancet.'⁴ In Holmes' 'System of Surgery,' Mr. Lee⁵ says, that it "is rarely, if ever, met with in females," while Mr. Johnstone⁶ adds to the accounts

¹ 'Surgical Pathology,' New Sydenham Society's translation, vol. i, p. 409.

² 'Surgery, its Principles and Practice,' ed. ii, p. 371.

³ 3rd ed., vol. ii, p. 174.

March 23, 1872.

Vol. v, p. 215.

Vol. iv, p. 35.

which I have already given, that "in some few cases it is much more severe; the cartilages become involved and permanent rigidity may be the result."

In a previous volume of these Reports¹ Dr. Pye-Smith describes the disease as a subacute and then chronic synovitis, confined to men, from which recovery is slow but certain. He notices that there is in most cases moderate œdema, and occasionally a slight inflammatory blush, but except for "the nocturnal exacerbation of pain," he knows no "symptom which points to implication of fasciæ or other fibrous tissues."

Mr. Erichsen, on the other hand, appears to have in view a very different class of cases, in the somewhat obscure account which follows.² "Gonorrhœal rheumatism principally occurs in young and otherwise healthy persons. It is of two kinds; in one, the most common, and indeed the typical variety, the fibrous and muscular structures are affected; in the other the joints are implicated. It is a disease that appears to be closely associated with some form of blood-poisoning, possibly in some cases with pyæmia. It is of two kinds, the fibroid or plastic, and the suppurative. The fibroid is intimately associated with those forms of blood disease in which fibrinous exudations are found in internal organs, more especially on the serous surfaces, as the pleura, the peri- and the endocardium. The fibroid or plastic form is not unfrequently accompanied by inflammation of the testicle or of the sclerotic. It commonly affects the fleshy parts of the body, as the hip, the shoulders, and the thighs, and not unfrequently occurs in the soles of the feet. It is always very painful at night, but is not accompanied by any very severe constitutional disturbance. The suppurative, more rare, appears in truth to be a variety of pyæmia, directly due to self infection of the patient from pus retained in the deeper portions of the urethra, or contaminating the system through the medium of the lymphatics. In these cases the inflammation, when it affects a large joint, is always monarthritic. The knee is one that specially suffers, permanent ankylosis consequent on destruction of cartilage resulting. The synovial form presents the ordinary characters

¹ Series iii, vol. xix, p. 341.

² 'Science and Art of Surgery,' vol. ii, p. 388;

of severe and often destructive inflammation of the joint, the knee or ankle being chiefly involved. Ankylosis is the chief danger to be apprehended as a remote result of gonorrhœal arthritis, and it is mostly incurable."

It would seem from the above that the cases in which Mr. Erichsen has observed this disease in joints, have all been of the suppurative form; but as, in his description of the treatment, he advises blistering and mercurial ointment for the local joint affection, I suppose that he has sometimes met with cases of simple hydrarthrosis.

The majority of these accounts appear to treat of the more chronic forms of gonorrhœal rheumatism.

The cases which I recorded in the 'Obstetrical Journal,' and those which I shall record in this paper, are of a more acute character.

This acute form is quite as often found in the female as the male, perhaps more often. It usually begins before the gonorrhœa or discharge has existed for any considerable time, and the subjects are generally under the middle age. The onset is like that of an attack of acute rheumatism. The patient is feverish, and has pain, tenderness, and swelling of several joints. These symptoms are often so severe that he has to take to his bed. In a few days the inflammation leaves all the joints save one, in which it concentrates itself with great severity. This may be any one of the larger joints; I have notes of its occurrence in the knee-, ankle-, wrist-, and elbow-joints, as well as in those of the carpus and tarsus. Most frequently I have seen it in the elbow. The appearance of the joint is not at all like that which is seen in acute synovitis. There may be effusion within the synovial sac, but the most striking characteristic is the œdema of the soft parts round the joint, accompanied, as a rule, during the height of the attack by redness of the skin. The swelling is in some cases very great, and when the elbow has been affected, I have seen the redness and œdema extending from the shoulder to the wrist-joint. I believe that the superficial effusion is due to the inflammation having attacked especially the fibrous tissues of the capsule rather than the synovial membrane by which it is lined. The slight amount of effusion which occurs in the synovial sac is probably secondary to the affection of the adjacent fibrous tissues, just as we

sometimes see an acute periostitis near the end of the diaphysis of a long bone accompanied by some effusion into the neighbouring articulation. The joint is very tender, hot, and full of pain, and any attempt to move it gives rise to excruciating pangs. The general temperature of the body is but little elevated. Perhaps a better idea of the condition of the parts will be conveyed if I mention the diseases with which this affection is likely to be confounded. I have several times seen it mistaken for simple or phlegmonous erysipelas, and I can recall one instance in which an incision was made into the reddened and inflamed tissues of a wrist when thus affected, under the impression that they were infiltrated with pus. There was a copious flow of blood, but fortunately the parts healed again satisfactorily. The great œdema which accompanies this form of inflammation sometimes suggests the presence of phlebitis, but the diagnosis is not difficult to make, as there is an entire absence of tenderness and cord-like induration along the track of the larger veins.

Gouty inflammation occasionally resembles it, but the youth of the patient would make us hesitate to ascribe the affection to this cause. Moreover, in gout there is usually a history of previous attacks or of hereditary predisposition, or of the habits which conduce to this condition. Lastly, I have had some cases brought under my notice with the diagnosis that they were suffering from chronic pulpy inflammation of the synovial membrane, on the point of breaking down into a general disorganisation of the joint, and the operation of resection has been suggested as the only treatment which afforded a prospect of recovery.

The prognosis of these cases is on the whole favorable. I have never seen suppuration occur, although sometimes the skin has been so red, hot, and tender, and fluctuation so distinct, that it has seemed almost certain that matter was forming. In every case, however, I have seen all these symptoms subside, with no worse result than more or less rigidity of the affected joint.

I have had no opportunity of examining a joint which has been the seat of this form of inflammation: but from the firm resistance offered to passive movement, and from the roughness of the motion, after this resistance has been overcome, I should

think that there is a plentiful development of fibrous adhesions and that the cartilages are often considerably eroded.

One point of great interest, as showing the alliance of this disease with acute rheumatism, is the occasional association of cardiac complications. In Case 3 it will be seen that there was on admission distinct evidence of pericardial inflammation, and in Case 6 of my paper in the 'Obstetrical Journal' it was noticed that there was a systolic bruit audible over the base of heart. Our own museum contains two if not three specimens of valvular disease associated with gonorrhœa.¹

In the first of these, however, there had been rheumatism one year previously, and the patient died during an attack of chorea; so the heart affection may not have been due to the gonorrhœal discharge. In the second there was no rheumatism. The third case seems to bear more resemblance to those recorded by me, though the report does not mention any evidence of gonorrhœa. It was from a single woman, aged 29, who died three months after child-birth with ulcerative endocarditis, and inflammatory lymph in the knee-joint. The inflammation of the joint came on after confinement, and is described as puerperal synovitis. Seeing that it was probably set up by the lochial discharges, and possibly by gonorrhœa, I think it may be fairly cited as an example of this association of cardiac inflammation with arthritis and purulent discharge from the genital organs.

In the article which I have already quoted, Dr. Pye-Smith mentions a patient with chronic gonorrhœal synovitis who was also the subject of a systolic basic *bruit*. At the same time, however, he states that he knows no "clear case on record of fever or endocarditis depending on gonorrhœal rheumatism." Continental observers, however, have recorded many such cases, and an analysis of thirteen, all occurring in men, will be found in the 'Revue des Sciences Médicales.'²

After I had written the foregoing account of this acute form of gonorrhœal rheumatism, I was much pleased to find that my observations were confirmed and supplemented by an interesting article contributed by MM. Duplay and Brun to the 'Archives Générales de Médecine.'³

¹ Preparations 1405⁶⁰, 1413⁶¹, and perhaps 1413⁶⁰.

² 1879, vol. i, p. 657.

³ " Sur une forme particulière et encore imparfaitement décrite d'Arthrite

Their description of this affection is very similar to that which I have given. The articulations in which they have found it more frequent are the elbow and wrist, but they have also met with it in some of the smaller joints, *e.g.* the sternoclavicular, and the phalangeal joints of the hand. They lay great stress upon the pain which accompanies and precedes the swelling and they remark that this pain is especially severe in the plane of articular contact. In certain joints also the tenderness is very acute at particular spots, *e.g.* in the wrist-joint just below the styloid processes, and in the elbow along the course of the lateral ligaments. They describe carefully the widely diffused œdema and swelling, and add that "in typical cases of acute blenorhagic arthritis, the idea of diffused phlegmon, of severe lymphangitis is the first to occur to the mind, and this idea agrees well with the general symptoms which although sometimes absent, are most often sufficiently pronounced, and with the rosy colouring, sometimes a bright red, of the integuments." They consider that "profound alterations of the ligamentous structures" are indicated by the abnormal movements, which may frequently be communicated to the joints, and that the articular cartilages are often attacked in the later stages of the disease.

Of the cases published by them, three are in males, and three in females, and all were the subjects of purulent discharge from the urethra. They do not consider that the acuteness or abundance of this discharge had any influence in the determination of this form of gonorrhœal rheumatism.

With regard to the treatment of the affection, I have not found any benefit from the use of the ordinary remedies for rheumatism. I have tried in vain the bicarbonate of potash, colchicum, iodide of potassium, salicylic acid, and other drugs of this class.

It is necessary to keep the patient in bed, and to put the limb in a splint. Warm applications of some anodyne lotion relieve the pain, and I have also found benefit from the use of an ointment composed of Ung. Hydr. Comp., mixed with extract of belladonna. As soon as the violence of the inflammation has somewhat abated, the pressure of a Martin's india-blennorrhagique," par MM. Duplay et Brun, 'Archives Générales de Médecine,' 1881, p. 541.

rubber bandage will be serviceable in reducing the swelling, at the same time that it lends a grateful support to the relaxed tissues. It is also very important to cure the discharge, which has set up the joint affection. Whether this be the result of gonorrhœa, balanitis, leucorrhœa, or any other cause it must be treated by the remedies appropriate to these affections. MM. Duplay and Brun speak very highly of the results obtained by the application of a plaster-of-Paris splint in these cases, and I shall certainly make a trial of it on the first opportunity. They say that in the majority of cases the pain disappears the same day, and that the swelling of the parts is rapidly diminished; and that where it has been soon resorted to, even in acute cases, it has usually led to complete restoration of the movements of the joint.

As there is little or no fear of suppuration, it is advisable, as soon as the swelling and heat have gone, to adopt such measures as may avert the fibrous ankylosis, which is so apt to ensue. Shampooing and passive movement are the best means of attaining this end, but where the disease has been acute, and the patient has come under proper treatment only after it has existed for a long time, it will generally be followed by a considerable rigidity of the articulation. I am disposed to think that a large number of the patients who come under us with stiff joints, the result of inflammation without suppuration in adult life, have been the subjects of this form of disease.

CASE 1. *Acute gonorrhœal inflammation of elbow, diagnosed as phlegmonous erysipelas; recovery.*—J. B—, æt. 19, lighterman, was admitted into Inner Samaritan Ward under me on May 20th, 1878. He had always enjoyed good health with the exception of an attack of gonorrhœa. Ten weeks before admission, after considerable exertion in rowing, his arm became very stiff, so that for a short time he could not use it. He noticed nothing particular about the elbow at this time. It gradually grew better, but after rowing a race on March 23rd, he noticed lumps in the right axilla with severe pain in the arm and forearm. After rubbing the limb with liniments, it got well, but a few days later, three weeks before his admission, his arm and the upper part of his forearm again became swollen, red, and painful. He attended as an out-patient, but as his

arm continued to grow worse he was admitted with what was described as a phlegmonous inflammation round the elbow. There was much swelling and redness of the soft parts; and he could not bear the least movement of the joint. His temperature was 101°. A mixture was ordered him containing persulphate of iron and quassia.

May 23rd.—The redness and swelling have subsided. The movement of the elbow is still painful, especially rotation; and there is fluctuation over the head of the radius. About this date I ascertained that the patient was suffering from gonorrhœa. An ointment consisting of 1 part of Ext. Belladonnæ to 4 of Ung. Hydr. Comp. was applied to the elbow, which was fixed by a splint. Subsequently he was directed to use an injection of Lotio Zinci Sulphatis (gr. j to the ℥j). On June 4th he was transferred to the Venereal Ward. The swelling and pain of the elbow gradually diminished, and on June 19th he went out.

It is said in the report that this patient was well when he left the hospital. I believe, however, that there was some, but not much, impairment of the movements of the elbow.

CASE 2. *Gonorrhœa; inflammation of right carpus; recovery.*—Mary A. C—, æt. 22, a cook, and unmarried, was admitted into Patience Ward under me on April 16th, 1880. Her family history was good, and she had always enjoyed good health. Six weeks before she had first noticed vaginal discharge and scalding during micturition, two weeks after coitus. For three days she had suffered from swelling of the right wrist.

On admission there was great pain and tenderness about the right carpus with considerable heat and redness. There was fair movement of the wrist and the lower radio-ulnar joints. She was unable to move the fingers, which were, however, but little swollen. She stated that she was suffering from vaginal discharge, but none could be seen upon examination. There was, however, considerable tenderness of the genital organs, and in subsequent examinations discharge was observed. An anterior splint was put on the forearm and hand, and an ointment was applied consisting of belladonna extract and Ung. Hydr. Comp. For a long time suppuration of the carpus appeared to be imminent.

On May 10th the measurements round the palm and wrist were 9 and $8\frac{3}{4}$ inches, as compared with $7\frac{3}{4}$ and 6 inches upon the unaffected side. At first she took salicylate of soda, then iodide of potassium, and latterly a simple tonic mixture containing cinchona bark and sulphuric acid.

By May 28th the wrist had become smaller and less tender. A shorter splint was applied, which allowed free movement to the fingers.

June 4th.—Extension of wrist through 10° ; flexion through about 30° . Fingers somewhat stiff.

8th.—Splint removed, and patient allowed to get up.

16th.—There is still some scalding during micturition.

July 2nd.—Fingers still so stiff that she cannot flex them to within half inch of the palm. Still some scalding and vaginal discharge.

12th.—She went out, with some stiffness of the wrist; her vaginal discharge had not quite ceased.

During the period of more than nine years, in which I took charge of patients in the female venereal ward, I do not remember another instance of similar joint affection, although I had numerous cases of vaginal discharge and gonorrhœa under my care. It would, therefore, appear that prostitutes are singularly exempt from gonorrhœal rheumatism, which seems, at least in the acute form, to attack with especial violence young married women, or those who without becoming prostitutes have been infected by an illicit connection.

CASE 3. *Acute inflammation of elbow; pericardial rub; balanitis; recovery.*—James S—, æt. 20, a leather shaver, was admitted into Philip Ward under Dr. Habershon, on April 28th, 1880. He gave a history of lumbago in his father, morbus cordis in his mother. Six months before he had suffered from pain in the wrists, but was able to work when they were bound up. Three days before he came to the hospital severe pain came on in the left wrist.

On admission he was suffering from inflammation of the left wrist. There was also a pericardial rub replacing the first sound over the mitral, tricuspid, and pulmonary areas. His temperature rose frequently in the evenings to 102° . On May

2nd the pericardial rub was not so distinct, but there was a systolic bruit; he was sweating profusely. On May 7th the left elbow had become considerably swollen, and the skin over it was glistening. About this time I was asked to see him, as it was thought that the elbow was about to suppurate. I found him thin and anæmic, and suffering great pain from his left elbow which was swollen and acutely tender. There was some redness of the skin above the olecranon. The left wrist was also tender, but neither red nor swollen. From the condition of the elbow, I at once suggested that it was due to some affection of the genital organs, and upon examination I found that he had phimosis, and that there was a copious discharge of pus from the orifice of the prepuce.¹ There was no scalding. A splint was applied to the elbow. Mist. Copaibæ was given and an injection of Lotio Potassæ Permanganatis ordered. In four days the discharge disappeared. The elbow was then less swollen and painful, the skin desquamating, but the soft parts were still œdematous. A few days later I could perceive some fluctuation in front of the head of the radius. Rotation of the forearm was limited, and some adhesions gave way with a distinct creaking sensation. The limb was then still kept in a splint.

May 17th.—Elbow still very œdematous. He was ordered Pot. Iod., gr. v, ter. die. This had to be discontinued on the 20th on account of a rash which had come out on his skin.

31st.—I manipulated the elbow gently and broke down some adhesions. An elastic india-rubber bandage was now applied.

June 6th.—Allowed to get up.

21st.—Quite well with the exception of a little thickening.

24th.—Use of the joint nearly returned.

28th.—Arm quite well and free from pain.

29th.—Went away to a convalescent home.

¹ I was informed that when the prepuce could be drawn back it was found that the discharge was due to a balanitis and not to any affection of the urethra. I cannot say from personal observation that this was the case, as I only saw this patient at considerable intervals. The second visit I paid to him the discharge had quite ceased. It seems to me not improbable that the balanitis was really the result of a gonorrhœa, although he stated that he had had no sexual intercourse for three months, and that in the interval there had been no discharge.

CASE 4. *Acute inflammation of right elbow ; vaginal discharge ; pregnancy ; abortion ; recovery with impaired movement.*—Eliza Y—, æt. 17, a collar-maker and unmarried, was admitted into Charity Ward under my care on August 18th, 1881. She was quite well until seventeen days before admission, when she noticed pain and swelling in her left hip. These went away, but reappeared in the right knee. Lastly the right elbow became inflamed, while the other joints recovered. For a few days the swelling of the elbow has been going down, but the stiffness has increased.

On admission she was healthy-looking and fairly nourished. She lay with her right elbow flexed to a right angle, unable to move it, and any attempt to flex or rotate the forearm made her scream with pain. There was moderate swelling of the elbow, the circumference of which was 10 inches, while that of the other side was $8\frac{1}{8}$ inches. The soft parts were puffy, but the skin was not discoloured. It was very tender and felt hot to the touch, but there was no rise in the general temperature. No fluctuation could be felt.

The œdema and excessive pain led me at once to attribute the inflammation to some affection of the genital organs. She said that her catamenia had ceased two months, having been previously regular, and that she had a white discharge. No vaginal examination was made, for there appeared to be no reason to suspect a venereal cause, and she denied to the sister of the ward the possibility of her being pregnant. She was ordered to use *Lotio Aluminis Comp.* as an injection, to take salicylate of soda in fifteen-grain doses three times a day. A splint was applied to the elbow and an ointment composed of belladonna extract and *Ung. Hydr. Comp.*

September 6th.—She aborted of an ovum, the size of a hen's egg. The elbow was somewhat better.

19th.—*Pil. Doveri gr. v, quartis horis.*

23rd.—The adhesions of the elbow were broken down under an anæsthetic.

28th.—She was allowed to get up.

October 4th.—*Lin. Camphoræ Comp.* ordered.

6th.—Range of flexion and and extension from 100° to 105° , rotation through about 15° .

26th.—She went out. The swelling had nearly gone, but

there was a good deal of firm tissue still surrounding the joint. During the last month passive motion had been employed to prevent the adhesions reforming.

When I last saw her on November 7th, she still had a scanty vaginal discharge, she could rotate the joint well, and the range of flexion and extension was from 90° to 130° .

It is singular that of the nine cases of joint inflammation in women, recorded by me in this paper and in the 'Obstetrical Journal' at least five were pregnant. At first I was disposed to attribute the affection to the pregnancy, but on further consideration it has appeared to me that the association of the rheumatism with pregnancy might be explained in the following way. As I have before said, nearly all my cases have been in recently seduced girls or young married women, and a considerable proportion of these would naturally be pregnant. We should, therefore, expect that unless pregnancy diminished their liability to the joint affection, the same proportion of those who had gonorrhœal rheumatism would also be pregnant. Perhaps, however, the fact of the discharge being accompanied by pregnancy, may render the patient more liable to the rheumatism.

CASE 5. Acute inflammation of elbow-joint; gonorrhœa and primary sore; recovery with some stiffness.—G. S—, æt. 19, labourer, was admitted into Luke Ward, under my care on December 22nd, 1881. About eleven weeks before, he one night felt his elbow very painful, and in the morning he was unable to move it. In two or three days it became swollen. No other joints were affected. There is no history of gout, rheumatism, nor phthisis in any member of his family. Does not remember to have had a blow or any kind of injury.

It was thought that he was suffering from pulpy disease of the synovial membrane, and that excision would be required.

On admission, the elbow was bent at an angle of about 105° , the forearm being half way between pronation and supination. There was considerable swelling of the soft parts, and a semi-fluctuating sensation could be felt especially at the back of the joint. The tenderness was most marked between the head of the radius, and the posterior border of the ulna. Active move-

ment was barely perceptible. Passive movement gave a good deal of pain.

Range of flexion and extension was from 95° to 110° or 115° ; of rotation through 20° .

The circumference of the right elbow was $10\frac{7}{8}$, of the left $9\frac{5}{8}$ inches.

The aspect of the joint, which resembled the subacute condition following an acute attack of gonorrhœal arthritis, led me to inquire whether he had recently had any venereal disorder. He then told me that three weeks before the elbow became inflamed, he had had a gonorrhœa, and that six weeks before his admission a chancre had appeared on the prepuce. On examination I found an indurated chancre on the right side of the prepuce more than half an inch in diameter. There was also a faint roseolous rash on the trunk. The fauces were normal.

He was ordered Mist. Hydr. Perchlor. \mathfrak{zj} , ter. die, and a Martin's india-rubber bandage was bound round the swollen elbow.

On January 16th the chancre was nearly well, and I find no report of its condition subsequently, but I believe that it was quite healed shortly afterwards, and that no other evidences of secondary syphilis developed themselves.

On February 2nd the swelling had nearly disappeared, but the movement of the elbow was as bad as on admission, if not worse. I therefore broke down all the adhesions under ether, and was able to move the joint thoroughly. The movements, however, were rough, as if the cartilage had been eroded or partly covered with fibrous tissue, and there was some looseness of the joint, probably from relaxation of the lateral ligaments.

On the 15th he went out. My last recorded examination of the arm was nine days previously. I could then move it fairly well, but the movement was rough, and I believe that at that time there was no power of active movement.

I am indebted to my colleague, Mr. Symonds, for permission to publish the following report. Knowing my interest in these cases, he kindly showed me the patient at the time when her elbow presented a most characteristic example of this form of inflammation.

CASE 6. *Acute gonorrhæal rheumatism of elbow; synovitis of knee; vaginal discharge; recovery with impaired movement of elbow.*—Lily T—, æt. 27, a barmaid, and unmarried, was admitted under Mr. Symonds into Patience Ward on May 17th, 1882.

No family history of rheumatism. About four years ago she had a sore between the labia, which got well of itself in a month or so. There is also an indefinite history of a vaginal discharge more recently. About three weeks ago she felt a stiffness in the shoulders, knees, and elbows, which she attributed to getting wet. There was then no swelling. The stiffness soon passed off, except from the left elbow and right knee, which became painful and swollen.

On admission the left elbow is swollen, and the skin somewhat red; it is œdematous and hot. The œdema extends some distance on either side the elbow-joint. The skin has a number of white lines upon it, as though it had been much more distended. Its circumference is 11 inches, while that of the right side is $9\frac{1}{2}$ inches. The depressions on either side of the olecranon are obliterated. There is considerable pain on movement and on pressure, and some pain while the arm is quiet.

The left knee is also slightly swollen and painful, and measures 13 inches as compared with $12\frac{1}{2}$ on the right side. There is a little fluid in the joint, but no redness nor œdema about it.

Temperature not above normal. She acknowledged that she had had a vaginal discharge recently, but as she stated that she had no trouble on admission, no examination of the vulva was made. Mist. Pot. Iod. \mathfrak{zj} , ter. die. An ice bag applied to elbow. The knee strapped with Ung. Hydr.

May 22nd.—The elbow is better, and measures only 10 inches in circumference. To be strapped with Ung. Hydr. The knee is much better.

June 1st.—I saw her. Her elbow was still somewhat swollen, hot and red.

17th.—Went out. The knee had been well for some time. The swelling of the elbow had nearly gone, and she could bear some movement. Her temperature had remained constantly below 100.4° .

When last seen at the end of July, all swelling had gone, but the movement was not complete. Passive motion was painful, and the patient was disinclined to persevere. She had a vaginal discharge which had increased since she left the hospital.

In conclusion I would suggest that there are several distinct affections confused together under the name gonorrhœal rheumatism. I would enumerate them as follows:

(1) *Gonorrhœal synovitis*, a chronic affection, occurring in the male, generally in the knee-joints. Very rarely it is acute, and goes on to suppuration. Of this kind I have seen no example, but cases have been reported to me which leave no doubt in my mind as to the possibility of this occurrence.

(2) *Gonorrhœal arthritis*,¹ an acute affection occurring in the female quite as often as the male, as a rule attacking at the outset several joints, and afterwards confined to one, most frequently the elbow-joint; affecting especially the fibrous tissues of the joint, and only secondarily the synovial membrane and cartilages.

(3) *Gonorrhœal inflammation of fibrous structures not connected with the joints*, e.g. the plantar fascia,² sclerotic, iris, the pericardium and endocardium. Inflammations of the three first structures usually occur in connection with chronic gonorrhœal synovitis. In the two last situations, as far as my very limited experience enables me to judge, inflammation is generally associated with gonorrhœal arthritis.

With respect to the second class, I would summarise my remarks in the following resumé:

1. It usually occurs during the acute stage of gonorrhœa or some purulent discharge from the genital organs, in adult patients under the middle age.

¹ I should prefer to use a word which should signify more clearly that the ligaments are the especial seat of inflammation. Perhaps Syndesmitis Gonorrhœica, or Gonorrhœal Syndesmitis, from *σύνδεσμος*, a ligament, would be the best term that could be adopted. The only objection I know to the word is that I am informed that it has been used as a synonym for Conjunctivitis.

² This gonorrhœal inflammation of the plantar fascia sometimes occurs in an acute form. My friend, Mr. Marsh, of St. Bartholomew's, informs me that he has seen flat foot rapidly produced on both sides by inflammatory softening of the ligaments of the side of the foot in a young man who was the subject of gonorrhœa.

2. It occurs as often in females as in males, if not more often. (Of the twelve cases recorded by me here and in the 'Obstetrical Journal' nine were females).

3. It may attack any joint, but most often the elbow (eight out of my twelve cases were in this joint).

4. At first it attacks several joints, like acute rheumatism, and then confines itself, as a rule, to one.

5. Its seat is the fibrous tissue of the joint. There is great œdema, redness, pain and tenderness. The ligaments are softened, the cartilage may be disorganised. There is very little synovial effusion. Constitutional disturbance is but slight.

6. It may be confounded at first with acute rheumatism, later on with phlegmonous erysipelas, bursitis, lymphangitis, phlebitis, gout, and pulpy disease of the synovial membrane.

7. It rarely if ever suppurates, but it is especially prone to set up fibrous ankylosis.

8. The best treatment is to cure the discharge, keep the joint perfectly still, and apply uniform pressure as long as the acute stage lasts, and then to use passive motion.

SOME REMARKS
ON THE
MINUTE ANATOMY AND ORIGIN
OF THE
ENCHONDROMATA OF THE SALIVARY
GLANDS.

By W. H. A. JACOBSON.

It is well known that new growths containing cartilage occur not unfrequently on the side of the face and upper part of the neck. The object of this paper is to supplement the somewhat scanty accounts which have hitherto been given of these growths, and to discuss the possible theories of the origin of the cartilage, a tissue which we should not have expected *à priori* to meet with in this region.

Position.—This may be (*a*) most frequently in the neighbourhood of the parotid, or, more exactly, just behind or in front of the angle of the jaw; sometimes they encroach upon the lobule of the ear or upon the tragus. (*b*) Much more rarely,¹ they are met with below the ramus of the jaw in connection with the submaxillary gland.

¹ Mr. Butlin, in the account of a specimen of Enchondroma of the Submaxillary Gland, brought before the Pathological Society, vol. xxviii, p. 228, alludes to the rareness of these tumours, his being the first case recorded in the 'Transactions.'

Sex in which these enchondromata are most frequently met with.—Out of twenty-four cases of which I have short notes, sixteen occurred in female patients. This great preponderance in women of this variety of enchondromata of soft parts appears to be in accordance with a statement of Prof. Virchow's, that certain cartilaginous tumours of soft parts, viz. those met with in the subcutaneous tissue, are relatively frequent in women, while cartilaginous tumours of bone predominate in the male sex.¹

Age.—These chondromata appear to occur most frequently between the ages of puberty and thirty.² Thus, of the above sixteen cases which occurred in female patients, twelve had not exceeded twenty-six years of age when the growth was first noticed; of the remaining four, in two the age is not given, and in the two others the patients were thirty-one and thirty-four respectively when the growth was first noticed. Of the eight male patients the new growth was noticed in four cases before thirty.

The new growths which, containing cartilage, occur in the neighbourhood of the salivary glands fall into the following two chief groups, which, however, are not separated by any hard-and-fast line from each other:

a. Pure enchondromata or fibro-enchondromata.—These, if small, consist of cartilage almost entirely, with very little fibrous tissue. If larger, they contain a greater amount of fibrous tissue, which invests, and, at the same time, separates the masses of cartilage, so as to ensure the presence of vessels.

b. Mixed enchondromata; "mixed parotid tumour."—The myxo-chondroma of Prof. Virchow. As these mixed enchondromata usually contain more or less perfect gland tissue (Pl. I, figs. 2 and 3), a yet more complicated name (myxo-adenochondroma) would be required to express their histological structure correctly.

External characters and aspect.—The enchondromata in relation with the salivary glands are usually firm and hard, somewhat tuberos, and often likened to a small potatoe or a bantam's or hen's egg. They are painless on pressure or after handling,

¹ 'Die Krankhaften Geschwülste,' Vor. xvi.

² Prof. Virchow, loc. supr. cit., draws attention to the frequency with which enchondromata, both of the bones and soft tissues, are met with during the first two decades of life.

with no adhesions to the skin unless treated by caustics; smooth at first, later on becoming more and more nodulated. This nodulation, or unevenness of surface, may in some cases be absent, and thus cause a difficulty in diagnosis, *i.e.* when the enchondroma lies deeply embedded in the gland substance, and when it fills its capsule tensely. The increase of these tumours is very slow and only inconvenient from the accompanying deformity. They do not interfere with the seventh nerve nor with the muscles of mastication, and cause no dryness of the mouth. Their mobility is often very slight. They owe this, even when quite superficial, to their position within the parotid capsule. When, as is often the case, they lie wedged in behind the ascending ramus of the jaw their increasing pressure gradually brings about considerable absorption of the surrounding gland tissue, so that, after the removal of one of these enchondromata, the surgeon may feel the styloid process lying exposed at the bottom of the wound.

The softer or more largely myxomatous variety of the enchondromata which are met with in this neighbourhood resemble the simpler form in many points of their position, painlessness, &c. But in proportion to the amount of soft or myxomatous tissue which they contain, they may be recognised more or less easily in different cases (*a*) by their more rapid growth—thus a history of two years instead of one of six or twelve may be given; (*b*) by the more elastic feel presented by different parts of the growth. This may be so pronounced as to resemble fluctuation owing to the displacement under the fingers of the mucin-containing fluid through the meshes of the myxomatous portion of the growth.

Very often, however, cases of soft enchondromata of the salivary glands which prove on examination to be large myxomata feel very firm to the finger before removal. This is owing to the tightness of their capsule, to the unyielding nature of the surrounding parts, and to the fact that these growths lie beneath a layer of parotid gland (p. 210).

Minute anatomy of the salivary enchondromata.—In this several distinct tissues have to be considered, viz. :—(*a*) The cartilaginous and the fibro-cartilaginous, (*b*) the glandular, (*c*) the myxomatous, fibro-myxomatous, and myxo-sarcomatous.

(*a*) *The cartilaginous and fibro-cartilaginous.*—This tissue

varies extremely, not only in different specimens, but also in different parts of the same specimen. It will be well to consider 1, the cells, and 2, the inter-cellular substance.

1. *The cells.*—These may be at one spot full in size and bulk, round, oval, or very bluntly angular in shape, but always in small and limited groups. Close by, and perhaps only separated by scanty intervening tissue, it is not uncommon to see groups of shrunken, exsuccous, withered cartilage cells. A third and distinct variety of cartilage is that with stellate cells, cells with irregular body-outline and numerous branching processes, suggesting either an attempt at conversion of cartilage cells into the lacunæ of bone, or, as I believe to be much more likely, recalling the relation of enchondromata to myxoma, another member of the connective-tissue family. Yet another distinct and very important modification is shown not unfrequently by the cartilage cells in these enchondromata; thus it is very common to find that they lose almost entirely the attributes of cartilage cells, viz. their rounded, oval, or angular outline, their capsules and capsule cavities, these being exchanged for cells which recall sarcomata from their rounded or spindle shape and granulation-like appearance; or else by delicate gradations becoming rounded and polyhedral, and gradually grouping themselves in more or less developed loculi which suggest gland-tissue, as in Pl. I, fig. 1. This specimen, which includes a portion of the new growth, its capsule, and the overlying salivary glands, shows how the cellular tissue of these enchondromata may consist very largely of little round, or angular cells of the “indifferent” or undifferentiated type, ready, as it were, to pass into tissue of a higher type whether glandular, cartilaginous, or sarcomatous. This large preponderance of elementary cell forms, which is so frequently met with in these tumours, and the readiness with which the above cell forms can be traced into higher tissue, affords, I would maintain, strong support of their embryonic origin, to which reference is made below, p. 215.

2. *The intercellular tissue.*—This, like the cells, shows great varieties. Thus at one place it will be homogeneous, transparent, or dimly granular (Pl. I, fig. 1, *d*). At another place the intercellular tissue will be fibrous or fibrillar; the former is best seen in the simple enchondromata, the bundles of

fibrous tissue chiefly serving to bring in a supply of blood to the otherwise evascular cartilage. Where fibrillar rather than fibrous tissue is present, the fibrillæ are often very delicate and tend to pass into myxomatous tissue (Pl. I, fig. 2). Occasionally they are coarser and recall the elastic variety. Well-marked reticular cartilage is, it is well-known, a rarity in new growths, but if my belief is well founded that these enchondromata originate in tiny masses of foetal cartilage which were found to be superfluous during the early development of the auricle, and which remaining latent and unnoticed for a while have at a later period of adult life begun to grow actively, then the reticular variety of cartilage, of which the human auricle consists, ought to be met with in these enchondromata of the salivary glands. Pl. I, fig. 4, shows, in the greater part of its periphery, cartilage of this variety, plump cartilage cells, oval and round, being seen to lie within a distinct reticulum.

b. The glandular.—This while usually present and giving rise in part to the compound title of mixed enchondromata, is often the least well-developed of the component tissues met with in any of these growths. Even where best marked the glandular tissue does not attain to a much higher degree than traces of a minutely acinous form; in other words, rounded or polyhedral cells with granular protoplasm and a distinctly staining, single, large nucleus are clustered together either in a circular or oval form, or else in that of tubes or cylinders. These acini, even when the glandular tissue in these new growths is best marked (Pl. I, figs. 2 and 3) are not often so grouped together as to reach the higher stage of lobulation. In other cases, as in Pl. I, fig. 1, *e*, *e'*, signs of acini are most rudimentary, the better marked glandular tissue being in these cases replaced by mere collections of simple, small, round, or oval cells, often ill developed and of nuclear rather than cellular appearance, and wanting entirely the glandular protoplasm or nucleus which has been alluded to above. These small, ill developed cells may be so arranged as to leave a distinct lumen (Pl. I, fig. 1, *e'*) or be so crowded as to quite fill up the rudimentary alveolus in which they lie (*e*). Further instances of the rudimentary nature of the gland tissue in these cases is shown (1) by the fact that a lining *membrana*

propria to the alveolus or acini is not always present, the gland-imitating cells lying in many cases, especially in the smaller groups, loose in the surrounding tissue, whether this be cartilaginous, fibrous, or myxomatous, (2) by the extreme rarity of any attempts at ducts, as indicated by tubes lined with columnar epithelium.

c. Fibro-myxomatous ; fibro-sarcomatous ; pure myxomatous ; pure sarcomatous.—It is by no means uncommon to find large portions of the “mixed parotid tumour” made up of delicate fibro-myxomatous tissue, and from the abundance with which this is studded with nuclei and rudimentary cell-forms the presence of sarcomatous tissue would be readily intelligible.

As these tumours, however, are usually removed, on account of the deformity which they cause, before they attain to any considerable size, the presence of any really pronounced sarcomatous element is very rare. It would appear that this element is present, but in a state of latent abeyance.

Pure myxomatous tissue is by no means so rare. From time to time we meet with softer varieties of the enchondromata of the salivary glands, though owing to the presence of a capsule and overlying parotid-gland tissue these softer tumours may feel as firm¹ as the commoner, hard fibro-enchondromata. When such a specimen, really a myxochondroma is cut across it usually consists of nodules of cartilage, but the intervening and connecting tissue which holds the nodules together is here not firm, fleshy, fasciculated, but semi-fluid or stringy. In its minute structure this tissue will be found to exactly resemble myxomata, *i.e.* it consists of irregular many-branched cells, the branches ending in fibrillæ which interlace so as to form

¹ This was very marked in a large parotid myxo-chondroma, which was lately sent to me by Dr. Fuller, of Shoreham. Before removal the growth felt so hard and nodulated that I expected it would prove chiefly cartilaginous, and I was inclined to doubt the man's history that it had existed barely two years. After removal, which necessitated division of the upper part of the sterno-mastoid, and during which the large vessels and the posterior belly of the digastric and the styloid process were exposed, the rapid growth of the tumour was explained by its being largely myxomatous, soft and even jelly-like in parts, though to the finger before the operation it had felt so firm. I believe that in this case owing to the presence of some congenital “moles” in the skin close by (p. 216) a nodule of foetal cartilage had long existed, which had recently taken on a more rapid growth of myxomatous tendency.

very delicate meshes, in a few of which may lie scattered cartilage cells.¹

While I believe such pure myxomatous tissue to form but a small part of the fibro-adeno-chondromata or usual "parotid tumour," and any pure sarcomatous element to be extremely rare, yet it is obvious from the rudimentary nature of the tissues which are met with in these tumours, and more especially from the embryonic character of the fibrous and intercellular tissue, and from the well-known relation of sarcoma to rudimentary or embryonic connective tissue, that these tumours contain an element of danger, and should thus always be removed early, quite apart from any accidental question of deformity. And this view perhaps gathers strength from the origin of these tumours which is suggested below (p. 215).

Removal.—This may be dismissed with a very few words. The main incision will of course be horizontal, and should be sufficiently free to enable the surgeon to get out the tumour which, if of long standing, may be larger than appears to be the case on examination. If the wound be made too small in the first case for fear of a scar, the edges will only be bruised and primary union prevented. It is not uncommon for branches of the facial nerve to be in relation with the capsule of the tumour, and if this has been much handled or treated by counter-irritation, they may very likely be firmly adherent, in either case injury to the nerve may be best avoided² by slitting up the capsule and shelling out the

¹ Mr. Arnott describes such a specimen of soft enchondroma of the parotid gland in the 'Path. Trans.,' vol. xx. Two admirable drawings are given, pl. ix, figs. 1 and 2. The patient was a man, æt. 23, but the duration of the existence of the new growth is not given. Though the non-cartilaginous part of the growth was softened, and "here and there, almost fluid so as to be capable of being drawn out in strings; at other places having the consistence and translucent flickering appearance of true colloid," the tumour, was, before removal, "very firm."

² If the surgeon, in making any deep incision that is necessary can manage not to go above the level of a line drawn horizontally three quarters of an inch below the lobule of the ear he will avoid any serious interference with the facial nerve.

Sometimes paralysis of the nerve, though marked at first, may begin to improve. Thus, in the case already alluded to (footnote, p. 210), where a large branch of nerve was dissected off the posterior and lower part of the growth, other branches

enchondroma first; the capsule should then be examined to see if any nerve branches are adherent to it, after these have been separated the capsule itself should be removed. This should always be done to prevent any recurrence, as the peripheral part of these enchondromata is often adherent to the capsule itself.

During the removal the substance of the gland should be interfered with as little as possible, for fear of causing leakage of saliva and a minute salivary fistula.

Origin of these enchondromata.—This matter would have appeared to have received hitherto very little attention. And this is the more strange, as the salivary glands are about the last place where the existence of cartilage in new growths, well marked and abundant, would *à priori*, have been expected.

The theories which may be put forward in explanation appear to fall under the following heads:

1. According to Prof. Virchow,¹ these new growths would owe their starting point to some "positive traumatic influence." He brings forward a case as evidence to show that in the etiology of cartilaginous tumours connected with bone contusions play an important part, but he mentions only one case in which a cartilaginous growth in the salivary glands followed an injury, in this case a cartilaginous growth in the parotid appeared to follow on a blow on the side of the face. Prof. Virchow's view is that an injury sets up a subacute inflammation of the gland which soon passes into a chronic interstitial parotiditis, the connective tissue being especially affected and showing signs of irritation, *e.g.* thickening, and microscopically, numerous small cells, fusiform in shape and indifferent in type, being scattered throughout the gland-tissue, or collected here and there into groups. From these "indifferent" cells, tissues of different kinds may spring, myxomatous, fibrous, and cartilaginous. Of these the last, with which we are most concerned here, may be hyaline or fibrous, or both these varieties of cartilage may be combined in the

being no doubt much pulled upon, considerable paralysis of the right eyelids, and to a less degree of the corner of the mouth, was present after the operation in January. Dr. Fuller writing two months later tells me that "the paralysis is much improved."

¹ 'Die Krankh. Gesch.,' Bd. i, Vor. xvi.

same specimen. Prof. Virchow believes that where the cartilage is about to be formed the "indifferent" cells increase in size, become rounded in shape, and surround themselves with capsules. If the intercellular tissue condenses itself and becomes homogeneous, hyaline cartilage will result; at another spot fibro-cartilage will be found, the intercellular tissue being occupied by fibres which have resulted from development in a different direction of the above-mentioned "indifferent" cells. At the spots where the inflamed connective tissue of the salivary gland is, according to Prof. Virchow, being thus developed into cartilage, the gland tissue itself is compressed and gradually wastes as the at first isolated nodules of cartilage, which are going to form the enchondroma, gradually appear. While this view, on account of the great authority of its supporter, should be mentioned first, it does not appear to me to be supported by clinical evidence; out of twenty-five cases of which I have notes, a blow is only mentioned in one. Furthermore, if I am correct in my belief that these enchondromata occur more frequently in women, this "traumatic" origin would, *à priori*, be less likely.

2. A few years ago Prof. Cohnheim¹ put forward an ingenious and suggestive theory and one which he supports by a profusion of suggestions and illustrations, that the main source of origin for tumours consists in certain relics of foetal tissues, which owing either to their being superfluous or to their development being arrested, have never reached maturity, but have remained quiescent in the midst of better developed tissues. The point at which the fault in development first arises is probably some early period of embryonic life when more cells being produced than are needed for the formation of the part, a certain quantity of cells remain unused, of very insignificant dimensions perhaps, but on account of the embryonic nature of the cells, of very great power of multiplication. Judging from Prof. Cohnheim's application of his theory to other cases, he would explain the origin of those enchondromata to which this paper refers in some such way as this. Some small heap of embryonic cells is left undifferentiated and undeveloped in the parotid; after some years, when the gland is in full functional activity, owing to the

¹ 'Vorlesungen über allgemeine Pathologie,' Berlin, 1877, S. 635, et seq.

vascular supply being abundant or on account of some slight injury the above relic of embryonic tissue takes on a fresh activity, and its undifferentiated cell elements are gradually developed into different tissues of the connective-tissue group, viz. cartilage, fibrous tissue, and as in Pl. I, fig. 1, by a grouping of cells on the plan of loculi into a rudimentary form of gland tissue.

One of the most brilliant of recent histologists, Dr. C. Creighton, in criticising Prof. Cohnheim's theory writes as follows:¹ "The objection that will be taken most widely to this hypothesis is that it is too exclusive. Whoever has at any time sought to attribute the origin of a tumour or tumours to a cause other than the proliferation of embryonic rudiments of tissue, will naturally feel that the hypothesis of Cohnheim is too wide. So comprehensive a hypothesis must needs swallow up many independent fragments of theory, as the rod of Aaron swallowed up the rods of lesser soothsayers. There is one great class of tumours for which the embryonic hypothesis is superfluous. It is not necessary to call in the aid of surviving embryonic rudiments to explain the tumours of secreting structures. A cause less remote, residing, in fact, in the everyday activity of such organs and parts, may be found for the tumours and diseases to which they are liable. In treating of the physiology and pathology of one secreting organ—the breast—I have endeavoured to explain its tumours as arising from disordered function,² and that explanation does not stand as an hypothesis, but as an induction. The rationale that is applicable to tumours of the breast may be applied also to tumours of other secreting structures. . . . If, then, the hypothesis of embryonic tissue rudiments be superfluous for the great class of tumours in secreting structures and other epithelial parts, it loses the unity or universality by which it stands or falls." Dr. Creighton then goes on to show by reasoning most ingenious, but, as it appears to me, here and there subtle and far fetched, that the principle

¹ "Illustrations of the Pathology of Sarcoma," 'Journ. of Anatomy and Physiology,' vol. xiv, p. 324.

² "Contributions to the Physiology and Pathology of the Breast," Lond., 1878; "The Infection of the Connective Tissue in Scirrhus Cancers of the Breast," 'Journ. of Anat. and Physiol.,' vol. xiv, p. 29.

of "disordered function" is applicable not only to new growths originating in epithelial structures, but also to those which start in tissues of the connective-type.

While Prof. Cohnheim undoubtedly intends to make his theory as comprehensive as possible, I do not gather that he would make it absolutely exclusive of all others. If so he would have, I think, but few disciples. In a field so wide, and one dealing with questions so complex as that of the etiology of tumours, all would probably agree that it is a mistake to shape one's faith by one creed alone. Dr. Creighton has proved, as far as inductive reasoning can prove anything, that in certain cases, *e.g.* carcinoma of the breast, the new growth takes origin in disorder of the function of the part. Prof. Cohnheim's theory appears to me to be especially applicable to quite another group, viz. the sarcomata, on account of their possessing many points of affinity to embryonic tissues.

I desire to point out in this paper that the region in which these mixed enchondromata occur is one which readily lends itself to support Prof. Cohnheim's theory, being one in which relics of foetal tissue might very naturally be expected to occur. To show the truth of the above statement we have only to consider (1) what takes place during the development of these parts, viz. the lower part of the face and the upper part of the neck, and (2) what is known to result when any step in the above development goes wrong.

1. At an early period, towards the close of the first month of foetal life, four buds or plates appear, called post-oral from the fact that they give rise to the parts behind and below the mouth. These plates fuse with their fellows of the opposite side in the ventral mesial line, but those on the same side are separated by intermediate branchial clefts, all of which close up in time save the upper part of the first which remains as the external meatus of the ear, the tympanum, and the Eustachian tube. In the first of these plates, which is known as "mandibular," runs a bar of cartilage, the cartilage of Meckel, in which, or, more exactly, in the membrane which covers its outer aspect, is developed the lower jaw. At the posterior and outer aspect of the first visceral cleft the auricle slowly appears as a kind of cutaneous operculum to the cleft. One other matter connected with the development of these parts

may be mentioned here as I shall briefly refer to it again (p. 217) below, viz. the fact that throughout foetal life the cavity of the tympanum is closed by very delicate connective tissue, which gradually atrophies as respiration is fully established.

2. Amongst the best known results which follow on disturbances in the above-described normal development are supernumerary auricles and fistulæ,¹ both aural and cervical. It is to the former, as bearing on the question of origin of the salivary enchondromata that reference will be made here. Supernumerary auricles may occur with or without fistulæ; when the two occur together they may be situated on different parts of the neck, or the supernumerary auricle, like a branchial operculum, may conceal the orifice of the fistula. Plate II gives several instances of supernumerary auricles as they occur without fistulæ. In fig. 1, which is taken from a patient of my own, the little nodule on the cheek showed distinct cartilage cells scattered irregularly through delicate fibrous tissue. It is noteworthy that the mother of this child showed a fistulous depression at the upper and anterior part of each helix. Figs. 2, 4, and 5 are copied by permission from patients which were under the care of Mr. Bryant. The central figure is from a patient of Mr. Birkett's, aged seven.

"The growths were situated over about the centre of the sterno-cleido-mastoidei muscles.² To the touch they resembled the lobe of the auricle, and they contained within them a firm resisting nucleus like the cartilage of the same organ. They were also covered with the peculiar delicate, soft, downy hairs like the lobe of the ear. I excised them without diffi-

¹ On this subject a paper by Sir J. Paget, "Cases of Branchial Fistulæ in the External Ears," *Med. Chir. Trans.*, vol. lxi, p. 41, may be consulted. Mr. J. H. Morgan has recorded two cases of Congenital Macrostoma, accompanied by malformation of the auricles and by the presence of auricular appendages in vol. lxxv of the same Transactions, p. 13. See also the following papers in Prof. Virchow's 'Archiv,' viz. "Hals-Kiemen-Fisteln von noch nicht beobachteten Fodern," by Heusinger, Bd. 29, S. 358, Taf. xii; "Ueber Missbildungen am Ohr und im Bereiche des ersten Kiemenbogens," by Prof. Virchow, Bd. 30, S. 221, Taf. vii, figs. 5—7; "Ein neuer Fall," von Halskiemenfistel," by Prof. Virchow, Bd. 32, S. 518, Taf. xii. In this case the ear is much deformed and the supernumerary auricle, situated immediately below it, is in a position in which enchondromata of the parotid are very frequently met with.

² *Path. Soc. Trans.*, vol. ix, p. 448. The drawing on pl. ii of this case is from a pencil sketch which Mr. Hurst has, and which he made at the time.

culty. Each was supplied with a small artery. They appeared to be intimately associated with the fibres of the platysma myoides, not dipping deeper than this structure, and to be entirely cutaneous appendages. A vertical section was made in the long axis of each growth, and the tissues of the lobe and of the fibro-cartilage of the auricle were clearly distinguished. The shape of the fibro-cartilage resembled more or less closely, in parts, the outline of that of the proper auricle, and its tissues were the same."

In the two figures (Plate II, figs. 4 and 5) the position of the fibro-cartilaginous nodule, corresponding most closely to the most frequent site of the parotid enchondromata, will not escape notice.

If, as these figures show, it is not uncommon to meet with, on the surface of these regions, these foetal relics in the shape of fibro-cartilaginous nodules or supernumerary auricles, it is not, I think, unjustifiable to suppose that similar minute masses of foetal tissue containing the elements of cartilage may occur not unfrequently embedded in the soft parts just beneath the surface, and that these little masses, which have failed to take their proper share in the development of the part, may, later on, form some of the salivary gland enchondromata.¹

In bringing this article to a close I would refer to the condition of the foetal tympanum. May not the fact that this is occupied with delicate connective tissue account for the origin and tendency to recurrence of some of the aural polypi. It is well known that many aural polypi which spring from the lining membrane of the tympanum and then perforate the membrana tympani, are myxo-sarcomatous in origin, that they tend to occur again and again unless entirely removed, and that their complete eradication is only to be brought about by perseverance on the part of both patient and surgeon. I would suggest that the structure and tendency to recurrence of these polypi may readily be explained on the supposition that they originate in some minute relics of that embryonic connective tissue which during foetal life has filled the cavity of the tympanum. If the above supposition be justifiable it affords one more instance of the applicability and of the correctness of

¹ On this point the case alluded to in the footnote, p. 210, may be referred to.

Prof. Cohnheim's view that a large number of new growths originate in remnants of foetal tissue which, not having been made use of during the development of the foetus, remain for a time latent and insignificant, but are always ready, from provocations or exciting causes, which are as yet unknown to us, to take on a pernicious vitality.

PLATE I.

FIG. 1.—This shows a section through part of a "mixed parotid tumour," together with its capsule, and the contiguous part of the parotid gland itself.

a. Parotid gland. *b.* Capsule of tumour. *c.* Peripheral portion of enchondroma in which crowds of round, oval, and oat-shaped cells of "indifferent" type are seen. In some cases this peripheral and growing portion of the tumour adheres intimately to the capsule. On this account the latter as well as the growth itself should always be extirpated. *d.* Cartilage. *e, e.* Here rudimentary acini are present, the undifferentiated cells, already alluded to, being collected into primitive loculi. In some cases the collections of cells lie in the surrounding cartilage without any attempt at a limiting membrane. (From a drawing by myself).

FIGS. 2, 3, 4 (from drawings by Miss Alice Boole) show other attempts at the formation of gland tissue. In Fig. 4 the periphery of the specimen is occupied by cartilage resembling that of the reticular variety.

PLATE II.

These figures are intended to show different forms of the congenital fibro-cartilaginous nodules which represent supernumerary auricles.

FIG. 1.—From a patient brought to me four years ago. The nodule on the cheek consisted of fibro-cartilage. The mother of the child had minute fistulae at the upper and anterior part of each helix. She stated that the same condition was present in her father's ears. The malformation of the auricle, which is not uncommon in these cases, is here very striking.

FIGS. 2, 4, and 5 are from drawings taken from two patients of Mr. Bryant's. They show well the pedunculated form which the above-mentioned nodules sometimes assume.

FIG. 3.—This represents a patient of Mr. Birkett's, whose case is related in the 'Path. Soc. Trans.,' vol. ix, p. 448. The position of the fibro-cartilaginous nodules is here quite below the level of the salivary glands. They probably originated in opercula to the second or third visceral clefts, and their presence here may perhaps explain the deep-seated fibro-enchondromata which are occasionally met with in the anterior triangles of the neck.

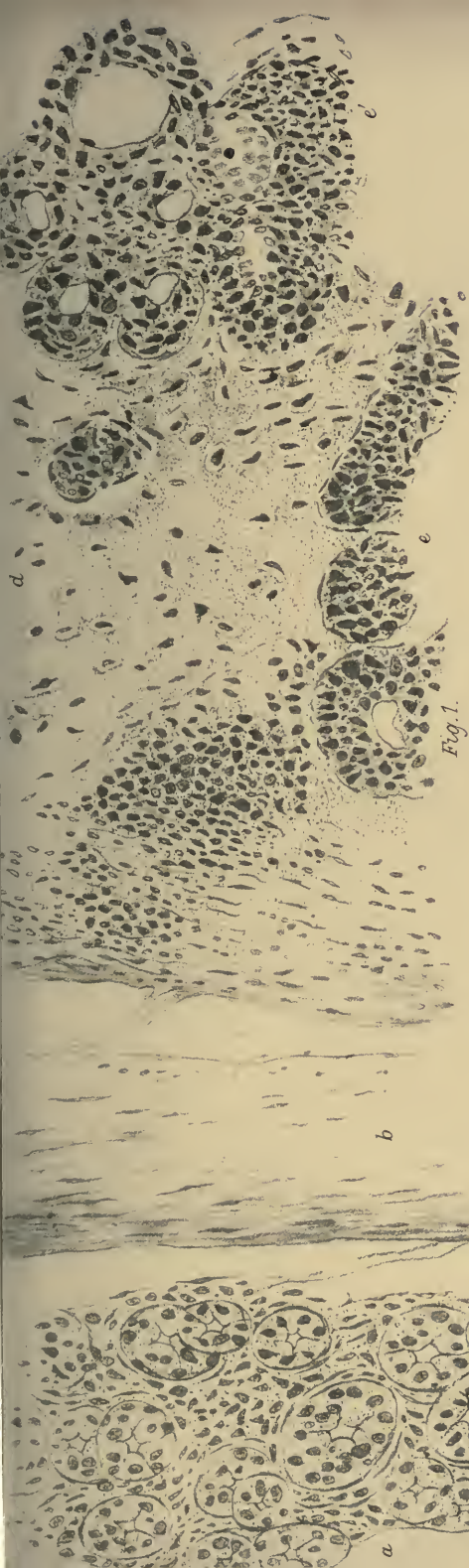


Fig. 1.

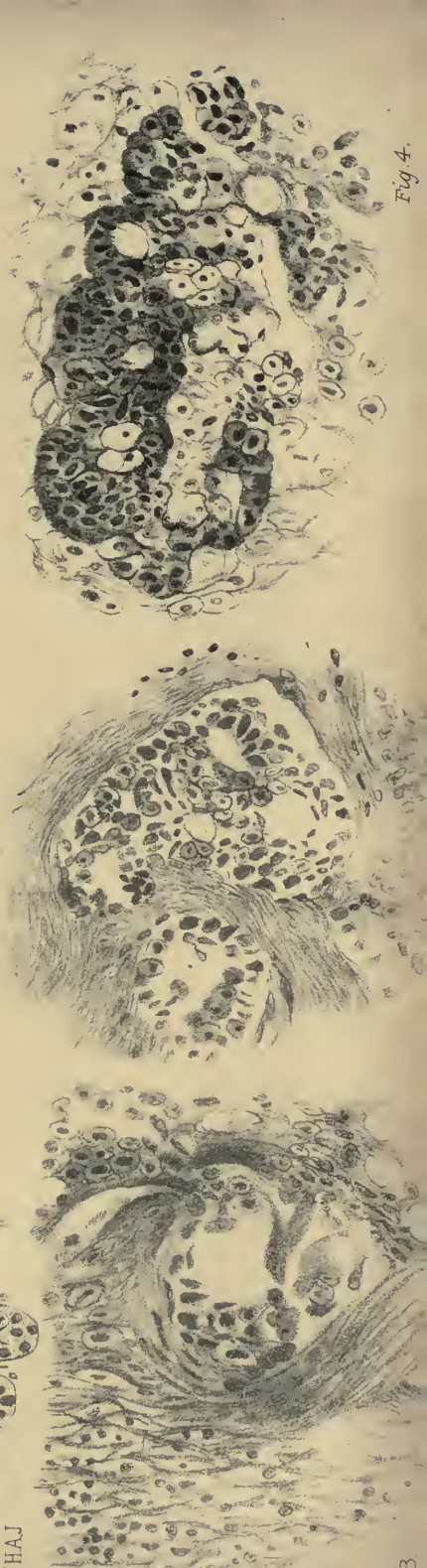


Fig. 4.



Fig 1.



Fig. 2.

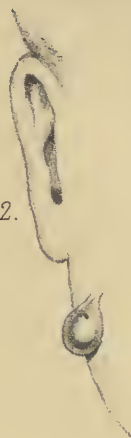


Fig 3.



Fig 4.



Fig. 5.



REPORT OF A CASE
OF
IDIOPATHIC ANÆMIA OF ADDISON,
SINCE CALLED
ESSENTIAL, PERNICIOUS OR PROGRESSIVE ANÆMIA.
WITH
A COMMENTARY AND TABLES OF SELECTED CASES.

By P. H. PYE-SMITH, M.D.

THE following case of a remarkable and obscure disease seems to find an appropriate place in these Reports, since it was within the walls of Guy's Hospital that the malady in question was first recognised and its characters defined. I have taken occasion to collect as many typical and completely recorded cases as I could find, and to give a critical account of the most important historical and pathological questions which are raised by a study of the subject.

The report is condensed from the full and accurate account drawn up by my clinical assistant, Mr. H. H. Sturge.

“ Thomas B—, æt. 46, a hatter by trade, living in the Old Kent Road, was admitted in the clinical ward under Dr. Pye-Smith, June 11th, 1879.

Family history.—His father died “ after a broken leg ” at seventy-two, his mother from “ dropsy and erysipelas ” at

sixty-four. Of six brothers, two have died : one at forty-seven of "diabetes and consumption," the other at forty-one of disease of the kidneys ; he had an apoplectic stroke three weeks before death. His only sister died from "water on the brain" when five years old. Two uncles and three aunts on the mother's side are alive and well. His father was an only child.

Personal medical history.—The patient is said to have been very weak as a baby ; the vaccination wounds did not heal for a year. As a boy he was fairly healthy, but about sixteen years ago he began to suffer from attacks of pain in the epigastrium and loss of appetite, sometimes with vomiting and always with a general sense of languor and depression, increased by food. These attacks have recurred ever since. Sometimes their duration has been two or three days, sometimes as many weeks. He is generally troubled in this way every month. He married at twenty-one, and two children by this wife are alive and healthy. Four years ago his wife died. He has since married again and has had two children by his second wife, the first stillborn. Nearly every summer since he was twenty he has suffered more or less from diarrhœa. About three years ago he contracted a chancre, which was not followed by sore throat or rash.

Present illness.—About a year ago he noticed an alteration in his complexion, and nearly at the same time his skin from head to foot became covered with "little blisters, like what are made by stinging nettles." This rash was attended with itching and stinging ; it lasted two or three days, and returned more than once within a fortnight. During the past year the dyspeptic attacks have become more frequent, with intervals of only a fortnight or three weeks, and have also been more severe. Although he thinks he has lost flesh before, he has become decidedly thinner only during the past year. Breathlessness on exertion appeared ten months ago and has increased.

He attended St. Bartholomew's Hospital under Dr. Duckworth's care for a short time.¹ He says that he some-

¹ His out-patient letter, dated March 8th, is headed "Anæmia," and contains the following prescription :

"℞. Pot. Iod., gr. vij ;
 Ferri et Am. Citr., gr. x ;
 Inf. Calumbæ, Aq. M. Pip., āā ʒiv. Ter die sum.
 "℞. Ol. Morr., ʒij. T. d. s."

what improved under treatment, but ceased to attend in consequence of a severe cold and cough, followed by diarrhœa.

On June 6th, 1879, he experienced a sensation as of a heavy weight at the stomach, and it has not left him; it is worse after food. He has suffered for six months from throbbing in the head, aggravated by exertion; palpitation and giddiness have accompanied this symptom. During the past year, too, he has had pains at times in the shoulders, hips, knees, legs, and ankles, brought on after exertion. No epistaxis or any other external loss of blood has occurred.

State on admission.—Patient says his height is five feet six inches and a half. Weight ten stone five pounds and a quarter. He says that some four years ago he weighed fifteen stone. He is well framed and fleshy, but the muscles of the limbs feel flabby. His expression is one of weariness rather than pain.

The skin all over the body is pallid, and has a yellowish tinge. The veins show clearly through it on the extremities. It is moist and warm and feels soft. The ankles are not swollen nor have they been so. On the palm of the left hand there is a dry, harsh patch, about the size of a halfpenny, partly covered with small shining scales, which do not come off on friction; it does not itch. There is a scar on the under surface of the glans and the prepuce.

Eyes.—Conjunctivæ very white, some œdema. On ophthalmoscopic examination the retinæ were found free from hæmorrhage.

Bones.—No irregularity except that the tubercles of both tibiæ are very prominent.

No *glandular* enlargement behind the sterno-mastoid, or in the axillæ or groins.

Some fulness of the *thyroid* may be observed.

Alimentary system.—Tongue pale, sodden, and tender. The lips, inside of cheeks, and gums are almost the same colour as the skin. On the left tonsil are two or three small yellowish patches, and a larger one on the right tonsil. Uvula and soft palate normal; but the parts are very pale. No dysphagia. Appetite very fair. Has not been sick for three weeks. Bowels act three or four times a day, motions loose. Liver dulness begins at sixth rib and does not extend below the thorax. No dulness in splenic region; for four inches

above margin of ribs on left side tympanitic resonance. Abdomen full and tympanitic.

Respiratory system.—Chest resonant all over. Vocal fremitus present at bases behind. At apices, an expiratory as well as an inspiratory sound is heard. Girth thirty-seven inches and a half. Sternum not prominent or depressed. No sinking in of intercostal spaces.

Circulatory system.—The cardiac impulse is seen below the sixth rib in a line with the nipple. The cardiac dulness does not extend to the right of the sternum; it begins at the third and ends at the sixth rib. A systolic murmur is audible at the third left intercostal space, and more loudly below and to the inner side of the nipple.

This bruit is also faintly heard at the ensiform cartilage. There is throbbing of the great vessels in the neck and below the left clavicle. Marked pulsation in abdomen about umbilicus, systolic in rhythm. Pulse 104, full, collapsing, compressible.

Urine.—Sp. gr. 1026, high colour, no sugar, no blood or albumen. Abundant salmon-coloured precipitate of urates.

Blood examined on the 16th.

Diagnosis.—Idiopathic anæmia of Addison (essential or pernicious anæmia).

June 11th.—Evening temp. 101° Fahr.

- ℞ Liq. Arsenic., ℥iij;
Aqua, ℥j. After food thrice daily.
- ℞ Ext. Nucis Vom., gr. ½;
Pil. Rhei Co., gr. iv. Every night.

Full diet with six ounces of wine.

12th.—Morning temp. 98·4°, pulse 90. Bowels open four or five times.

13th.—Evening temp. 99°. Bowels open eight times in twenty-four hours. Omit pills.

14th.—Evening temp. 99°, pulse 100. Bowels open once to-day. Pains less. Urine in twenty-four hours ℥xxiij.

15th.—Evening temp. 99·2°.

16th.—Evening temp. 99·4°. "Feels weaker." Sick last evening about one hour after the medicine; "he felt very exhausted for one hour after vomiting." Pulse 96, throbbing. Bowels open only once yesterday, the motion was loose but of

a natural colour. No pain in stomach. Finger pricked and blood examined; it was of a natural colour; counted white cells in three fields, numbers were fourteen, twelve, twelve; thought the cells did not form rouleaux well, and that there were present one or two small cells. No variety of form of corpuscles. From appearance of blood the disease is certainly not leucæmia.

17th.—Pulse 92, evening temp. $99\cdot7^{\circ}$. Bowels open once yesterday "Throbbing in head and aching much easier." Urine deep colour, sp. gr. 1035, contained 2·8 per cent. of urea, by Russell and West's modification of Davy's method.

18th.—Evening temp. $99\cdot6^{\circ}$. Urine Jxxliix , 2·8 per cent. of urea.

19th.—Morning temp. $99\cdot2^{\circ}$, pulse 100, resp. 16, evening temp. $99\cdot2^{\circ}$. Urine sp. gr. 1026, no albumen, deep red line on adding it to nitric acid at junction of the two fluids, 2·7 per cent of urea; urine in twenty-four hours Jxxxvj . Sat up yesterday evening, and says he feels better for it to-day. After walking a dozen yards and having his throat looked at with laryngoscope on 17th was quite exhausted.

20th.—Pulse 100, temp. $99\cdot2^{\circ}$, resp. 18. Feels no appetite for dinner to day. Yesterday after dinner began to feel sick, and the sensation continued until after tea when he vomited. "Slept pretty well." "More faint and languid" than yesterday. Lips externally are rather more red than on admission. No improvement in colour of tongue and gums. Bowels open every day. Evening temp. 99° .

R. Liq. Arsenic., mv ;
Tr. Camph. Co., Jj ;
Aquam, ad Jj . T. d. s., after meals.

Urine in twenty-four hours Jxxviiij , sp. gr. 1025, not such a deep orange colour as that of yesterday; bottom of utensil has been for last two days covered with red deposit.

21st.—Morning temp. $98\cdot4^{\circ}$. Feels better. Blood examined at 3 p.m.; the red corpuscles did not form rouleaux at all well; in some places after forty-five minutes the disks had lost their characteristic appearance; corpuscles were seen to be smaller than the ordinary discs (microcytes), and several were pear-shaped instead of circular. In four successive layers of blood, not more than one corpuscle deep, I counted the white

corpuscles, and found the numbers were six, sixteen, sixteen, and twenty-three. 9 p.m.—Temp. $99\cdot4^{\circ}$, resp. 16 (sighing), pulse 88. Says he feels “more depressed and languid than before.” Urine in twenty-four hours \mathfrak{Zxxxv} .

22nd.—Temp. $98\cdot2^{\circ}$, pulse 92, resp. 16. Has been thirsty and wakeful for last three days. Urine in twenty-four hours \mathfrak{Zxxxij} , 1·6 per cent. of urea.

23rd.—Morning temp. 99° , pulse 96. Getting out of bed while it was being made caused palpitation and much shortness of breath. At apex of heart a systolic bruit is heard, and a clear second sound. Over subclavian and axillary arteries a “grating” bruit is perceptible. In neck a pulsatile humming sound is heard; it is not connected with breathing, for when he holds his breath it is more plain. On placing the stethoscope between the xiphoid cartilage and the umbilicus and pressing, a soft bruit is heard with every pulse. Over femoral a throbbing sensation is conducted to the ear, but no distinct murmur. His abdomen round umbilicus throbs very plainly with each beat of the heart. Visible pulsation in both radials and over right thenar eminence. Blood looked at just before his dinner: white cells in two fields counted, numbers were seventeen and nineteen; the same small red disks and irregular-shaped corpuscles as before were observed. “No apparent decrease of red, certainly no increase of white corpuscles; rouleaux well formed, a few microcytes, but not more than in healthy blood. No nucleated red corpuscles, many pear-shaped, a few crenated, a few white corpuscles undergoing amœboid changes at ordinary temperature.” Evening temp. 99° . Urine in twenty-four hours \mathfrak{Zxxx} .

24th.—Temp. 99° , pulse 96, resp. 12. Feels better this morning. Has not taken medicine since yesterday evening. Now (11 a.m.) sweating, whole skin moist. Urine \mathfrak{Zxxvij} , sp. gr. 1025, clear.

25th.—Morning temp. $98\cdot8^{\circ}$, pulse 100. Blood tested by hæmochromometer. Blood drawn into small pipette up to ten. This quantity placed in tube and water added up to sixty. The fluid thus made does not equal that given as normal colour for an equal state of dilution by one third. Under the microscope four and six leucocytes respectively were counted in two fields. After six hours almost every cell was crenated,

and numerous filaments of fibrin had formed. Evening temp. 99.8° . Right eye looked at with ophthalmoscope: fundus extremely pale, disc pale, no hæmorrhages.

R Liq. Arsen., mvj ;
Tr. Camph. Co., ʒj ;
Aquam ad ʒj . T. d. s.

26th.—Morning temp. 98.4° , pulse 100. Urine high colour as usual, sp. gr. 1022, 2.2 per cent. of urea (by Apjohn's modification of Davy's method), ʒxl of urine passed in the twenty-four hours. Feels better.

27th.—Blood looked at on warm stage (Hartnack, No. 7 objective), five white cells seen undergoing amœboid movements. The changes took place very rapidly at first, so that the pencil was never more than ten seconds or so off the drawing paper. Fields chosen where corpuscles were not thick and the white cells counted, numbers were 3, 3, 6, 2, 6, 3. After ten minutes rouleaux were perceptible, and four hours after the corpuscles were either irregularly massed together or separate. Urine after standing thirty hours deposited a slight flocculent cloud of mucus in the lower half of the glass, but no sediment; microscope shows only one or two epithelial scales. 9 p.m.—Temp. 99.2° .

28th.—Evening temp. 99.3° . Urine ʒxxxviiij , 2.9 per cent. of urea.

30th.—Pulse 96, morning temp. 99.2° . Urine in twenty-four hours ʒxxx . Sphygmographic tracings taken: apex somewhat rounded, diastole marked but not excessive. "Decidedly better," "not so breathless."

July 1st.—Lifts sixteen pounds from the ground with comparative ease, using both hands. Raises eight pounds with right hand and can then extend the arm with the weight, but he gets breathless after one or two efforts.

2nd.—10 p.m.—Complains of pains in knees and hips. Evening temp. 98.6° , pulse 100, resp. (while asleep) 14. Urine ʒxxxviiij .

3rd.—Morning temp. 99.3° . Feels worse, "very weak." Evening temp. 99° . Blood again examined with hæmocytometer. The numbers of cells counted in ten squares were respectively 15, 10, 12, 11, 10, 23, 7, 13, 14, 9, giving an average of 12.4 for each square. To the blood of a healthy

person taken for comparison and equally diluted, 22 cc. extra of soda-sulphate liquid had to be added in order to bring the colour down to that of the solution of patient's blood. Urine ℥lvij .

4th.—Urine ℥lvj . Fowler's solution increased to ℥viiij three times a day.

5th.—Urine ℥l .

6th.—At base and apex of heart both sounds are heard, the first in both places being accompanied by a murmur. Bruits also heard over axillary, carotid, brachial, and subclavian arteries, not marked over aorta, none over femoral. Throbbing of aorta as visible as before, but patient is no longer troubled with the sensation of throbbing in the head which he had when he entered. General colour of body not altered. Urine ℥lvj .

7th.—Urine ℥xl . Venous hum heard in neck.

8th.—Evening temp. 99° . Complains of aching in both eyes and says this symptom has occurred now and then during the past four months. Belladonna ointment to be rubbed over eyebrows. Urine ℥xlvj ; sp. gr. 1017.

12th.—Urine ℥xliv .

13th.—Urine ℥lij .

14th.—Blood: few microcytes, not many white corpuscles, three counted in a field; the red disks form rouleaux well. Urine ℥xliv .

15th.—Weight (in same clothes as before) eleven stone two pounds. Urine ℥lx , sp. gr. 1018.

16th.—Urine ℥lxij .

17th.—Urine ℥xlvj .

18th.—Urine ℥xliv , lighter colour.

19th.—Blood again examined by hæmocytometer. In ten squares there were the following numbers of red disks: 15, 14, 14, 12, 14, 16, 20, 22, 20, 20, only two or three leucocytes seen in the whole number. Blood solution weaker in colour than that of health, so that ℥v of water had to be added to the latter before the colours of the two were the same.

21st.—The patient has much improved in colour; this is especially noticeable in the lips. Has no complaint. Urine ℥lxiv .

22nd.—Urine ℥lvj .

23rd.—Evening temp. 99.4° . Systolic murmur heard at base and apex, but it is not distinct. Bruits heard over carotid,

subclavian, axillary, and brachial arteries on right side. No murmur heard over aorta or either femoral. Abdominal pulsation still visible. *Bruit de diable* audible in neck.

26th.—Much stronger; he helps an atactic patient to walk up and down the ward, and gives him considerable support.

28th.—Blood again examined with hæmocytometer. In ten squares were the following numbers of corpuscles: red, 26, 25, 14, 17, 19, 15, 16, 19, 21, 20; white in five fields, 24, 11, 5, 5, 10. Taking the average of red disks in this observation, $19.2 \times 2 = 38.4$, the hæmal unit. Or $38.4 : 100$ normal as $1,920,000 : 5,000,000$. My own blood gives in ten squares these numbers: 46, 50, 47, 50, 45, 46, 44, 43, 37, 48, average 45.6 ; hæmal unit = 91.2 , i.e. 4,560,000 instead of 5,000,000 corpuscles per cubic mm.

29th.—Left to day. The same dose of arsenic has been regularly taken since July 4th. Colour returned to lips and to some extent to face; his aspect strikingly improved since his admission."

This patient, after leaving Guy's Hospital, continued taking his medicine in eight- or ten-minim doses thrice daily, and used to come to see me from time to time. He continued to improve remarkably, both in colour and in strength, and if he was unable to get a renewal of the medicine for a week always said that he felt the worse for the want of it.

At the close of the year he was readmitted into the hospital under Dr. Wilks, who kindly allows me to make use of the following notes by his clinical clerk, Mr. George Currah.

January 1st, 1880.—The patient says he continued to improve after he left the clinical ward; he gained strength and colour, and had a good appetite. In October he noticed a change in his condition; vomiting came on, he began again to suffer from shortness of breath, and his appetite failed. This change would last for a week and he then would improve a little, but every relapse caused him to feel weaker, and he noticed that he was becoming again very pale. He went on in this way till December 31st, when he came to Guy's and was readmitted into Stephen Ward, No. 29. The following notes were then taken:

The skin all over his body is extremely pallid, and has a yellowish hue. The conjunctivæ are quite white. The thyroid is not enlarged, and there is no undue prominence of eye-

balls. No enlarged lymph glands. Fairly nourished. Tongue white, inner surface of lips and gums very pale. Appetite bad. Has vomited occasionally for the last three months. Bowels regular, sometimes act two or three times a day. Liver and spleen normal; chest normal; no cardiac bruit can be heard either at base or apex of heart. A loud *bruit de diable* is heard on right side of neck, slightly on left. There is a moderate subclavian murmur. Urine sp. gr. 1025, no albumen, blood, or sugar, rather high colour. Pulse 100, temp. 99·8°.

5th.—Blood was drawn from tip of finger by a needle at 10.30 a.m. immediately after lunch, but too soon for this to have had any effect on the blood. When drawn it had a pale, thin, watery, transparent appearance. Hæmoglobinometer showed 75 per cent. of colouring matter absent. Hæmocytometer gave twenty-six as the hæmic unit, or 1,300,000 to the cubic millimètre instead of 5,000,000. The ratio between the red and white corpuscles was one white to 212 red. Examined on the warm stage of the microscope, the red corpuscles appeared mostly oval in outline, often crenated, some circular; size generally larger than usual, but some were only two thirds the size of normal red corpuscles: these were very few in number. The dark appearance produced by the concavity was not always in the centre. "Two or three appeared from their shape to be undergoing division, and I watched one while it divided into two."—G. C. The colourless corpuscles were as large or larger than those of normal blood, and showed active amœboid movements. Some, however, were not above two thirds of the size of normal leucocytes. Of these also a few showed movements, most were of a circular shape and motionless. Besides ordinary red and white corpuscles, there were small bodies observed, round or irregularly shaped, some of a yellow tint, others colourless; these showed no movements. Max Schultze's granular masses of protoplasm were looked for, but only two or three of these were seen.

7th.—Pulse 100, temp. 99·6°. The patient has had diarrhœa to-day, which has caused him to feel rather weak. His former medicine was renewed.

8th.—Feels better to-day; slight frontal headache.

9th.—Pulse 96, temp. 100°. Looks rather better, somewhat improving in colour.

10th.—Sleeps well, and says he is feeling stronger. Blood examined this morning with the following results:—The hæmoglobinometer showed 75 per cent. of hæmoglobin absent. By the hæmocytometer 25 per cent. of red corpuscles were present, or 1,260,000 to the cubic millimètre. Under the microscope the proportion of leucocytes was one to 550. On the warm stage the red disks were mostly oval in shape and crenated, not biconcave, and some very irregular in outline. The colouring matter was collected in an irregular shape either in the middle of the corpuscle or, more often, eccentrically. No rouleaux were observed. The larger leucocytes do not show such active movement as before, and their number is not so great. The smaller corpuscles, of round or irregular shape and yellowish colour, are not so numerous as before. Many granular masses.

11th.—Patient says he feels stronger; his colour is better, appetite good, he has no vomiting, and sleeps well. Temp. 98·8°.

13th.—Has had diarrhœa to-day, attended with a great deal of pain.

14th.—Feels better this morning. The blood was again examined with the following result:—Hæmocytometer showed no increase in number of corpuscles. On the warm stage the number of oval corpuscles was less, and that of round ones greater, than before; many were crenated. A great decrease was noted in the number of small, round, and irregular corpuscles.

15th.—Complains of a feeling of sickness soon after he takes his medicine.

16th.—Gets up in the evening, but feels rather weak. No return of diarrhœa.

19th.—Medicine omitted on account of nausea.

20th.—Patient feels better; was up for two hours last night.

21st.—Does not suffer from sickness.

22nd.—Medicine resumed—

R Liq. Arsen., miv ;
Tr. Opii, miv ;
Aq., ʒss . T. d. s.

23rd.—Patient is not so well to-day; complains of pains in head, eyes, and stomach, is very languid and drowsy.

24th.—Pains in head much worse and appetite not so good.

℞ Lig. Arsenic., mv ;
Aq., ʒss . T. d. s.

26th.—Appetite is improving ; he has a better colour, and says he feels stronger.

29th.—Is able to get up in the morning.

31st.—Continues to improve.

The quantity of urine passed daily during January was noted. It varied from one pint to two pints six ounces, usually between one pint and a half and two pints.

February 2nd.—Is getting stronger, but says he feels pain in his hip-, knee-, and ankle-joints.

3rd.—Complains of headache and pains in the stomach.

8th.—Seems rather better and lips not so pale.

9th.—Liq. arsenicalis increased to mvj for a dose.

10th.—Complains of headache and sickness. Medicine omitted.

12th.—Patient is not so well ; is paler, and still complains of sickness and pain in the stomach.

℞ Tr. Chlorof. Co., mxv ;
Aq., ʒj . T. d. s.

15th.—Is feeling better ; no return of sickness or headache.

16th.—Medicine resumed.

℞ Liq. Arsenic., miv ;
Tr. Chlorof. Co., mxv ;
Aq., ʒj . T. d. s.

20th.—Patient walked out in the grounds to-day. Colour better, appetite good.

25th.—Has severe headache and sickness together with diarrhœa. Medicine omitted. Enema amyli.

26th.—Diarrhœa stopped, but patient feels very weak ; has been suffering several days from pain in the stomach, which is increased after food.

March 1st.—The dyspeptic symptoms are gone.

℞ Liq. Arsenicalis, miv ;
Tr. Chloroformi Co., mxv ;
Aq., ad ʒj . T. d. s.

No pain or sickness, but paler again,

15th.—Weakness and pallor increasing, the arsenic was omitted though he continued free from dyspeptic symptoms. One of the phosphorus “perles” to be taken three times a day.

22nd.—The phosphorus has had no good effect. Patient says that he is weaker under this medicine than under the other, and that it causes his head to throb and makes him sleepy. Former treatment resumed.

℞ Liq. Arsenicalis, ℥v;
Tr. Chloroformi Co., ℥xv;
Aq., ad ʒj. T. d. s.

29th.—

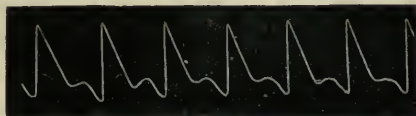
℞ Liq. Arsenicalis, ℥viij;
Tr. Camph. Co., ʒss;
Aq., ad ʒj.

April 17th.—The hæmocytometer showed 1,200,000 red corpuscles per cubic millimètre.

20th.—Venous transfusion performed by Mr. Bryant. The patient's son, a healthy-looking young man, furnished the blood. Before transfusion the following notes were made:—Apex beat directly in nipple line, two inches below it; first sound prolonged. Reduplication of first sound between nipple and sternum. Slight reduplication of second sound at apex. The hæmocytometer showed 700,000 per cubic millimètre. Under the microscope



Sphygmographic tracing taken before transfusion.



Sphygmographic tracing taken immediately after transfusion. It will be seen that there is only a minute difference in the apex of the tidal wave. The pressure was, of course, the same.

the corpuscles appeared of various sizes and of odd shapes (pear-shaped and elongated); many small, irregular-shaped granules were present in addition to the blood-corpuscles. Nearly four ounces (110 cc.) of blood were transfused from arm to arm by Dr. Aveling's instrument. Evening temp. 100.4° .

21st.—Morning temp. 99.8° , evening temp. 100.2° .

22nd.—Morning temp. 100.4° , pulse 108, full and bounding. Hæmocytometer showed 490,000 corpuscles per cubic mm. Evening temp. 99.8° . Feels comfortable.

23rd.—Morning temp. 99.4° , evening temp. 100.2° , pulse 112. Worse this morning; has vomited several times, and has had attacks of faintness during the night. No rigors. Hæmocytometer showed 490,000 corpuscles per cubic millimètre.

24th.—Relieved; but faintness and vomiting have not ceased. Morning temp. 100.4° , evening temp. 101° .

25th.—Sleepless, frequent vomiting, and faintness. Morning temp. 100.2° , evening temp. 101.4° .

26th.—Vomiting continues with occasional attacks of faintness. Pulse weak, 108, morning temp. 100.4° . Heart: at base both sounds reduplicated, apex beat very feeble and a little farther out—three eighths of an inch outside nipple. Evening temp. 100.8° .

27th.—Slept better last night after a chloral and bromide draught. Complains of palpitation. Pulse 108, temp. 99° . At 2.15 p.m., transfusion was again performed by Mr. Bryant. The operation was finished at 2.40; six ounces (187 cc.) were transfused. About 3 o'clock he began to breath laboriously, and soon after complained of being cold, and shivered. At 3.30 the rigors were severe, respiration laborious and deep, thirty-six per minute. He groaned constantly, and complained of pains all over his body. Temperature at 3.30, 101° , at 4, 102.2° . At 4.15 he felt better, and by 7 o'clock was pretty comfortable and breathing quietly. Evening temp. 99.4° .

28th.—Looks very yellow, but there is no jaundice. Has no pain and breathes quietly. Pulse 104, variable in strength. Apex beat in about the same position, a quarter of an inch outside nipple line. At apex, first sound is now altered to a

sharp, more valve-like sound, though between the nipple and sternum it still retains its bruit-like character; second sound still indistinctly reduplicated. At base, first sound sharper in character. Hæmocytometer gives only 530,000.

29th.—Pulse 105; has vomited two or three times. Nutrient enemata administered.

30th.—Feels better. Pulse and respiration improved.

May 1st.—Worse this morning. Complains of exhaustion and of aching pains all over. Respirations deep, pulse 114, bounding, very compressible. Vomiting returned. Mental faculties somewhat cloudy. Morning temp. 101° , evening temp. 100.2° . Ordered hydrocyanic acid in an effervescing mixture.

2nd.—Morning temp. 101.4° , evening temp. 101.2° .

3rd.—The legs are becoming œdematous and there is some puffiness of the hands and arms. Morning temp. 102° .

4th.—Morning temp. 101° .

5th.—Very drowsy. Heart's apex a little further out, and second sound more markedly reduplicated at apex. Morning temp. 100.2° , evening temp. 101° .

6th.—Morning temp. 99.6° , evening temp. 101° .

7th.—Morning temp. 99° , evening temp. 99.4° .

8th.—Continues in about the same condition, sometimes drowsy and apathetic, sometimes breathing deeply and hurriedly with symptoms of distress. He has general anasarca, the legs, arms, hands, and face swollen, with subconjunctival œdema. Morning temp. 99.6° , evening temp. 99° .

9th.—Morning temp. 99.4° , evening temp. 100° .

10th.—Eyes examined with the ophthalmoscope. No hæmorrhages seen; the whole retina, especially around the optic disc, was very pale. Morning temp. 98.2° , evening temp. 98° .

11th.—Much worse to-day, and suffering from dyspnœa. Morning temp. 99.4° , evening temp. 99.8° .

12th.—Last night he had severe diarrhœa with vomiting. Anasarca and dyspnœa have increased. Morning temp. 103° , evening temp. 103.4° .

13th.—Died this morning at 3 a.m. During the last few hours his breathing became still shorter and more difficult.

Sectio cadaveris (twelve hours after death).—On opening the body plenty of fat was seen to surround the internal organs. There was a little clear serous fluid in the chest.

Lungs pale, very œdematous, structure healthy, some old adhesions on the right side.

Heart rather large and flabby. It weighs $13\frac{1}{2}$ oz. Some patches of ecchymoses were seen on right auricle. The great vessels were of a bright vermillion colour, the endocardium of the auricles was also stained. No clot was found in the heart. The muscular tissue of the heart was very pale, and presented the "tabby" aspect of degeneration.

Kidneys.— $11\frac{1}{2}$ oz. Structure appeared healthy. One was excessively pale, the other was partly pale and partly stained, as if from decomposition of blood.

Liver.—63 oz. Very anæmic, but dark; not very fatty.

Adrenals.—Perfectly healthy.

Stomach.—Mucous membrane pale, coloured with bile.

Spleen.—9 oz. Very soft; on section the pulp had the appearance of a soft jelly.

Brain.—48 oz. Very pale, vessels thin. No ecchymoses of substance or of meninges.

Eyes.—No retinal hæmorrhages were present.

Bones.—The vertebræ, humerus, sternum, tibiæ, and ribs were examined and sections made, but nothing abnormal could be found either by the naked eye or by the microscope.

The above full and, I fear, tedious account, gives a fair picture of the disease first recognised by Addison and described by him in the Introduction to his last and most widely-known contribution to pathology, the short treatise 'On the Constitutional and Local Effects of Disease of the Supra-renal Capsules.' I will quote the passage at length.

"As a preface to my subject, it may not be altogether without interest or unprofitable to give a brief narrative of the circumstances and observations by which I have been led to my present convictions.

"For a long period I had from time to time met with a very remarkable form of general anæmia occurring without any discoverable cause whatever, cases in which there had been no previous loss of blood, no exhausting diarrhœa, no chlorosis, no purpura, no renal, splenic, miasmatic, glandular, strumous, or malignant disease.

"Accordingly, in speaking of this form in clinical lectures,

I, perhaps with little propriety, applied to it the term "idiopathic" to distinguish it from cases in which there existed more or less evidence of some of the usual causes or concomitants of the anæmic state.

"The disease presented in every instance the same general character, pursued a similar course, and, with scarcely a single exception, was followed after a variable period by the same result.

"It occurs in both sexes; generally, but not exclusively, beyond the middle period of life; and, so far as I at present know, chiefly in persons of a somewhat large and bulky frame, and with a strongly-marked tendency to the formation of fat.

"It makes its approach in so slow and insidious a manner that the patient can hardly fix a date to his earliest feeling of that languor which is shortly to become so extreme. The countenance gets pale, the whites of the eyes become pearly, the general frame flabby rather than wasted; the pulse perhaps large, but remarkably soft and compressible, and occasionally with a slight jerk, especially under the slightest excitement. There is an increasing indisposition to exertion, with an uncomfortable feeling of faintness or breathlessness on attempting it; the heart is readily made to palpitate; the whole surface of the body presents a blanched, smooth, and waxy appearance; the lips, gums, and tongue seem bloodless; the flabbiness of the solids increases; the appetite fails; extreme languor and faintness supervene, breathlessness and palpitations being produced by the most trifling exertion or emotion; some slight œdema is probably perceived about the ankles. The debility becomes extreme; the patient can no longer rise from his bed; the mind occasionally wanders; he falls into a prostrate and half-torpid state, and at length expires. Nevertheless, to the very last, and after a sickness of perhaps several months' duration, the bulkiness of the general frame and the obesity often present a most striking contrast to the failure and exhaustion observable in every other respect.

"With perhaps a single exception, the disease, in my own experience, resisted all remedial efforts, and sooner or later terminated fatally.

"On examining the bodies of such patients after death I have failed to discover any organic lesion that could properly

or reasonably be assigned as an adequate cause of such serious consequences ; nevertheless, from the disease having uniformly occurred in fat people, I was naturally led to entertain a suspicion that some form of fatty degeneration might have a share at least in its production ; and I may observe that, in the case last examined, the heart had undergone such a change, and that a portion of the semilunar ganglion and solar plexus, on being subjected to microscopic examination, was pronounced by Mr. Quekett to have passed into a corresponding condition.

“ Whether any or all of these morbid changes are essentially concerned—as I believe they are—in giving rise to this very remarkable disease, future observation will probably decide.

“ The cases having occurred prior to the publication of Dr. Bennett’s interesting essay on “Leucocythæmia,” it was not determined by microscopic examination whether there did or did not exist an excess of white corpuscles in the blood of such patients.

“ It was whilst seeking to throw some additional light upon this form of anæmia that I stumbled upon the curious facts which it is my more immediate object to make known to the profession.”

Then follows the account of the disease since known distinctively as *Morbus Addisonii*, or, as the author called it, *Melasma suprarenale*.

We have here the clinical characters of idiopathic anæmia portrayed—its course, its termination, and the post-mortem results stated. The facts are communicated as something hitherto undescribed, and a sufficiently distinctive title is given. This was no attempt to discover a new disease in a single case. The writer was well known as one of the ablest physicians in England, who was remarkably reticent in his statements and only too unwilling to appear in print. He was writing shortly before the close of his distinguished career, after more than thirty years’ experience in one of the largest hospitals in London. Addison had taught the existence and clinical features of “idiopathic general anæmia” in his lectures, he referred to it in the paper in which he first drew attention to *Melasma suprarenale* in 1849,¹ and it was recognised by his

¹ In a paper read before the South London Medical Society on “Anæmia and

colleagues in Guy's Hospital and by other London physicians. Thus, in the volume of these 'Reports' for 1855, published shortly before the description above quoted, Dr. Wilks speaks of the absence of leuchæmia "in that class of cases which has specially gained the attention of Dr. Addison, and which he has designated *idiopathic anæmia*." The same writer, in his 'Lectures on Pathological Anatomy,' published in 1859, describes (p. 459) these cases of fatal anæmia, with the bloodless condition during life, the œdema of the lungs and other parts, the passive serous effusions, the pale blood with its want of coagulation, and the fatty degeneration of the heart. One of the cases recorded in 1863 (No. 12) by another pupil of Addison, Dr. Habershon, was cited by Potain among the references at the end of his article "Anémie," in the 'Dictionnaire Encyclopédique des Sciences Médicales' (1864), and several other cases of the new disease were published both in England and abroad before 1870, as will be seen by the table B, at the end of this paper.

When, however, Prof. Biermer's papers appeared in 1871, the disease was accepted in Germany as a new discovery, and the name he proposed of "progressive pernicious anæmia" has been adopted by Immermann, Quincke, Hermann Müller, and Eichhorst. Neither Biermer, nor Immermann, nor Scheby-Buch in his collection of cases of "Essential Anæmia with Fatal Result"¹ seem to have heard of Addison's work. But on the first report of the "new disease from Germany," Dr. Wilks stated the facts of the case in a letter to the 'British Medical Journal' (November 28th, 1874), and some months before Dr. Scheby-Buch's paper appeared, two cases were published in perhaps the best known periodical in Europe, with a quotation of Addison's original description and a vindication of his priority.²

Disease of the Suprarenal Capsules," published in the 'Medical Gazette' for March, 1849. This shows, however, "truth in the making," for the author had not yet clearly separated what we now call idiopathic anæmia from what we now call morbus Addisonii.

¹ "Zur Casuistik u. Literatur der essentiellen Anämie mit tödtlichem Ausgang," 'Deutsches Archiv f. Klin. Med.,' Bd. xvii, April, 1876.

² "Zwei Fälle von Anaemie progressiva perniciosa," 'Virchow's Archiv,' Bd. lxy, p. 507, December, 1875. These cases are mentioned by Scheby-Buch, but apparently he had only seen the title of the paper.

In 1877 M. Lépine, in the January number of the 'Revue Mensuelle de Médecine et de Chirurgie,' blames Prof. Biermer for having neglected the researches of Addison, but quotes several cases of fatal anæmia recorded by still earlier writers. Some of these are also recorded by Scheby-Buch. A few are undoubtedly genuine cases, as will be seen by a conspectus of them in the table (A) at the end of this paper. Dr. Barclay's two cases published in 1851, two of Lebert's in 1854 and 1858, probably also Piorry's in 1840, and Marshall Hall's case of fatal chlorosis may claim a place. Many such might be found. One I have tabulated, which is extremely well described, both in the symptoms and the post-mortem appearances, by Dr. J. S. Combe and Dr. Kellie in 1823.¹ In the same year a case of Andral's was published which M. Lépine thinks may have been idiopathic anæmia; but it probably

¹ As this case is probably the earliest on record I give an abstract of the full and interesting account in the 'Transactions of the Medico-Chirurgical Society of Edinburgh' for 1823, p. 194.

The patient was a man, æt. 47, healthy and well nourished, who "had never been blooded." In July, 1821, he complained of weakness and dyspnoea on exertion, but for two or three months before his complexion had become pale. He was then seen by Dr. Combe, who describes him as "exactly resembling a person just recovering from an attack of syncope. The face, lips, and whole surface were of a deadly pale colour, the albuginea of the eye bluish." Pulse 80. Bowels irregular; thirst and anorexia. Urine pale and copious, with scarcely any sediment. Feet and eyes œdematous. In September he tried a sea voyage and drank chalybeate water, but returned home in October with loss of flesh and anasarca, the anæmia continuing as profound as before. In January, 1822, he died with all the symptoms of hydrothorax, having passed the last twenty-four hours of his life in a state of lethargy. A *post-mortem* examination was made by Dr. Combe and Dr. Kellie, who adds a few particulars in the course of another paper in the same volume (p. 119). The body was nearly the same colour as it was during life; no hypostasis; little rigor. All the organs were healthy, though excessively bloodless. There was œdema of the pia mater, and serum in the pleura and pericardium. The heart was pale, "like flesh macerated many days in water." No mention is made of tabby mottling of the muscle, nor were any ecchymoses noted. The spleen was soft, the kidneys bloodless, the great vessels empty.

Dr. Combe quotes a case from the 'Miscellanea Curiosa' of *exsangue fere corpus*, and refers to Lieutaud's account of "anæmia chlorosis," and also to an endemic anæmia which in 1802 affected more than fifty workmen in a coal mine near Valenciennes. May this have been due to the same disease which produced similar results among the workmen in the St. Gothard tunnel, the *Anchylostoma duodenale*?

anticipated the researches of Bright rather than those of Addison.

After Biermer and Immermann had made "pernicious anæmia" celebrated in Germany, Prof. Lebert (then of Breslau) put forward a claim to priority on the strength of the above-mentioned cases and some later ones, which he had called "puerperal chlorosis," and had described both in French and German during his professorship at Zürich. In his '*Grundzüge der ärztlichen Praxis*,' 1868, he speaks of essential or idiopathic anæmia ending, in rare instances, in death, when no organic disease can be discovered. The three cases recorded in 1854 of "a peculiar chlorotic condition, acute, febrile, and coming on quickly after delivery, were cured with steel in a few weeks." The single case published in 1858 was fatal, and may have been what Addison would have recognised as fatal idiopathic anæmia.¹

There can be no doubt of the independent value of Lebert's observations, but even if the genuine character of Combe's and Piorry's cases be questioned, Dr. Barclay's are indisputable and are earlier than Lebert's first cases; while Addison's description of a group of cases, which may be fairly called "a new disease," was earlier as well as more comprehensive in 1855 than Lebert's in 1868 or Biermer's in 1872.

I venture to think that the facts adduced prove that to Addison belongs the credit of recognising and describing this remarkable morbid condition. The work of Biermer and Immermann was no doubt perfectly original, and does credit to the acumen of these eminent physicians, but their conception of the disease was more narrow and perhaps less accurate than that of Addison. Moreover their path had been indicated by the previous observations of Lebert and of Gusserow in their own district. Nor does this rectification depend upon obscure or forgotten records, or upon isolated cases imperfectly described and half understood. It is almost always possible to rake up such cases, since really new diseases, like osteitis deformans, are at all events very rare. If we look for such examples of so-called pernicious anæmia we find them in English

¹ In a note on p. 2 of the Introduction to his work on '*Pernicious Anæmia*,' Dr. Hermann Müller criticises Lebert's claims in a way which compares with M. Lépine's criticism of Biermer.

as well as in continental literature. But Addison published a complete account of the disease as something new, he named it, and he taught his colleagues and disciples to recognise its characters. It was included by Dr. Wilks in the first edition of his well-known lectures nearly ten years before Biermer's earliest publication, and cases were published from time to time as instances of the disease. Dr. Barlow, in his 'Practice of Medicine,'¹ quotes Addison's description and adds: "to these cases Dr. Addison states that he applied the term idiopathic anæmia."

Nor was this knowledge confined to the medical school of this hospital. Dr. Bright and Dr. Quain were familiar with the facts. At St. Thomas's Hospital Dr. Bristowe, at the London Hospital Dr. Sutton,² and in Canada Professor Howard,³ had taught the same doctrines as Dr. Wilks was teaching at Guy's.

Assuredly there is no desire to extenuate the important additions made by subsequent observers abroad to the facts observed by Addison. They receive, I trust, due recognition in the summary which follows. Nor can the vast debt which medical science owes to Germany be diminished by rendering justice where it is deserved.

It was the lot of Addison to "stumble upon," to use his own modest expression, no less than four morbid conditions before unrecognised or imperfectly described: xanthelasma, described by himself and Dr. Gull under the name vitiligoidea; keloid, or morphœa, which, as Dr. Fagge has shown, is closely allied to, or identical with, sclerodermia; idiopathic anæmia; and melasma suprarenale. But his fame does not rest on these more or less complete discoveries: Addison's greatest work was the series of brief but masterly papers in which he put forth his doctrines on pneumonia and on phthisis, and anticipated some of the most important principles of modern pathology.

¹ 2nd. Ed., 1861, p. 542.

² "Thanks to the teaching of Dr. Sutton, and to Dr. Wilks calling attention to Addison's description, I have been familiar with the disease since a student, and have seen characteristic cases from time to time" (Mackenzie).

³ "Dr. Howard, of McGill University, has long taught the existence of Addison's idiopathic anæmia" (Drs. Gardner and Osler).

Leaving the question of priority, we will now trace the progress made in definite knowledge of the disease by observation and record of cases.

The first addition to Addison's facts was made by Dr. Wilks, who, in a collection of cases from the wards of Guy's Hospital (Nos. 1—6 in Table B), states that there is no increase of white corpuscles in the blood. This appears to have been the first microscopic examination made of the blood in idiopathic anæmia, and was no doubt suggested by the then recent discoveries of Virchow and Bennett.

Lebert's paper, "*Ueber Essentielle Anämie*," appeared a year later (in 1858), and two remarkable cases by Dr. Bristowe and the late Dr. Leared in the '*Pathological Transactions*' for the same year (Nos. 8 and 9 in table). Dr. Bristowe remarks (p. 437) that probably death was "due to severe and progressive anæmia. . . . If the view here suggested be correct, the case is allied to those examples of anæmia to which Dr. Addison alludes in the commencement of his work on diseases of the suprarenal capsules."

Passing over two well-recorded cases (Nos. 10 and 12) from Guy's, and one (No. 13) from St. George's Hospital, we come next to a passage in the '*Clinique Médicale*' of Trousseau. In the nosologies of Cullen and of Thomas Young, chlorosis appears as a species of dyspepsia; and, influenced possibly by this conjunction, the eloquent professor of the Hôtel Dieu describes a case of "*cachexie anémique*" (No. 15 in the table) at the end of his lecture on dyspepsia. There was no leuchæmia, but the account of the autopsy is too scanty to make one sure of the nature of the case:—"La rate était volumineuse, tendue, dure, comme hépatisée; les glomérules de Malpighi étaient évidemment augmentés de volume. . . . Le cœur était petit et très-anémique." Possibly this case may have been one of anæmia splenica, like one which I recorded in the '*Pathological Transactions*' for 1870 (p. 390). A similar case appears in Mosler's '*Pathologie und Therapie der Leukämie*' (p. 188).

In 1860 Cazenave published a case in the '*Journal de Médecine de Bordeaux*,' which I have seen only in abstract. It seems to have been genuine idiopathic anæmia proving fatal in a young man of twenty-one (No. 11 in Table).

Perroud's cases, published at Lyons in 1865, were regarded by him as anæmia and dropsy dependent upon fatty degeneration. Thus one is headed "polystéatose viscérale," and another, "stéatose du foie; appauvrissement du sang, cachexie et anasarque." This connection had been previously recorded, as we have seen by Wilks, and the true sequence more correctly appreciated. One of Perroud's cases seems to have been latent enteric fever and another fatty degeneration from alcohol. Two others I have included in Table A.

A case was published by Corazza, of Bologna, in 1869, of "oligæmia," with remarks on its connection with chlorosis and with fatty degeneration (No. 37). The patient was a woman, twenty-four years old, who was exhausted by frequent pregnancies and privation. She had all the signs of profound anæmia without emaciation, and after death nothing was found but effusion in the pleuræ and fatty degeneration of the liver and kidneys.

In 1871 Prof. Gusserow, now of Strassburg, published five cases of "hochgradigste Anämie Schwangerer." They were all fatal, and, except enlargement of the spleen, no organic disease was found. In two of these cases transfusion was tried. They were all observed at Zürich. I have included two in the table (Nos. 19 and 20); the others were cases of sudden death after labour, and may be compared with those in a paper by my late friend Dr. Phillips, which appeared in these 'Reports' for 1873.

In 1868 Prof. Biermer (then of Zürich) had made a communication to the "Versammlung deutscher Naturforscher und Aerzte," when it met at Dresden, entitled "Eine vorläufige Mittheilung über fettige Degeneration des Herzens und der Gefässe in Folge von Anämie." Four years later, in the 'Correspondenzblatt für schweizerische Aerzte' for 1872, he announced as a new discovery the existence of a condition which he named "progressive perniciöse anämie." He then stated that he had observed many such cases since 1867 and recorded fifteen; these were all among poor Swiss peasants, mostly women between twenty and fifty years of age. The condition was occasionally idiopathic, more frequently it began in privation, dyspepsia, or diarrhœa, rarely in chlorosis. The spleen was not enlarged and there was no evidence of

malaria. He described the yellowish pallor without jaundice, the maintenance of subcutaneous fat with progressive anæmia, the œdema, the muscular weakness, palpitation, anorexia, cardiac and vascular murmurs, and occasional pyrexia, the almost constant fatal result, and the fatty degeneration of the heart with only bloodlessness and œdema of other organs. All this was described just as Barclay, Lebert, Addison, Wilks, and other writers had described it before. One new and important observation Prof. Biermer made, that among the organs liable to hæmorrhage in the disease was the retina, and thus was added a valuable help to diagnosis during life.¹

In 1874 Prof. Immermann, of Basle, published two cases of "progressive pernicious" anæmia. Only one was verified by a post-mortem examination (No. 21 in table). With them appeared a remarkable third case, which was correctly diagnosed during life as leuchæmia myelogenica. Decided increase of white corpuscles was found on three occasions; and post-mortem there was not only hyperplasia of the marrow in the sternum, ribs, thoracic vertebræ, and femur, but also great hypertrophy of the solitary and agminated follicles of the intestine. This case, therefore, forms a connecting link between those of myelo-leuchæmia described by Neumann and Mosler, and those of ileo-leuchæmia recorded by Heschl and Béhier. In this paper Immermann defines the chief characteristics of the "new disease" (as he supposed it to be) as follows:— (1) Absence of preceding hæmorrhage or other cause; (2) extreme degree of the anæmia; (3) pyrexia without local inflammation; (4) the progressive and malignant course. It is to be regretted that in so well-known a work as Ziemssen's 'Handbook' the article on so-called "Pernicious Anæmia" by Prof. Immermann entirely neglected the work of physicians who preceded Prof. Biermer and himself.

In the same year (1875) I published a short paper with two cases in Virchow's 'Archiv,' and took occasion to translate the most important part of Addison's original description of the disease.

"A Contribution to the Casuistics of Pernicious Anæmia"

¹ Prof. Biermer's cases are numbered 48 to 68 in Table B, including others recorded by his assistant Dr. H. Müller. I have added the dates of the earliest in square brackets.

by Zenker, in the same volume of the 'Deutsches Archiv' as Immermann's paper (xiii, p. 348), is a case of anæmia as the result of uterine hæmorrhage, with mitral endocarditis and other complications, and shows the confusion introduced by the use of "pernicious," instead of "idiopathic," "essential," or "primary," as the designation of anæmia.

Prof. Quincke's cases in Volkmann's 'Sammlung' (1876, No. 100) and the 'Deutsches Archiv' were (like those of Lebert, Gusserow, Biermer, and Immermann) taken from among the peasants of Switzerland. He does not, however, mention Lebert, and the only English case he quotes is that reported by Dr. King, and numbered 16 in the table at the end of this article. One of his cases was complicated by dysenteric colitis, one by atrophy of the stomach, and one by a chronic abscess of the lung; one recovered, and another proved to be enteric fever. Four are included in my table. Like Lépine, Rosenstein, and other observers, Quincke found the liver, pancreas, and other organs stained of a dark colour and rich in iron, probably from the medicine given, but *perhaps* also from destruction of hæmoglobin. In other respects, the occasional pyrexia, the retinal hæmorrhages, the fatty degeneration of the heart, Quincke's description agrees with that of previous observers. He accepts the title "pernicious" as not excluding occasional recovery, but objects that the course of the disease is not always "progressive."

Lépine's paper already referred to ("Sur les Anémies Progressives") appeared in 1877. He quotes Addison's description and disinters from earlier writers isolated cases of fatal anæmia, which may, with more or less likelihood, be recognised as belonging to the same group. He would, however, extend the term essential anæmia, so as to include cases which depend upon recognised and more or less adequate causes, insufficient food, pregnancy, dyspepsia, and diarrhœa, and even those in which there is decided affection of the spleen, lymph-glands, or marrow. A review of Dr. Lépine's valuable paper, with a critical account of the cases published up to that time, appeared in the 'London Medical Record' for August and November, 1877 (pp. 314 and 437).

In the same year, Dr. Byrom Bramwell published an interesting account of eight cases of idiopathic anæmia, with tempe-

rature charts and a drawing of the blood-corpuscles. The two with autopsies are included in my table as Nos. 43 and 44. Two others, in sailors who had suffered from yellow fever, may possibly have been of malarial origin. But the chief interest of the paper is the apparent and remarkable success of treatment by arsenic in three of the cases.

Also in 1877, a volume of 250 pages was published by Dr. Hermann Müller, of Zürich. It is entitled 'Die Progressive Perniciöse Anämie,' and is dedicated to Prof. Biermer, whose wards furnished the majority of the cases described.

In the same year but later, appeared a valuable monograph, with the same title, by Professor Eichhorst, of Göttingen, based upon observations made while assistant to Prof. Frerichs at Berlin. He enters fully into the literature of the subject, and especially considers the claim to priority made by Lebert and on behalf of Addison. The former he justly disallows. With respect to the latter author, he admits that the idiopathic form of progressive pernicious anæmia was known before Biermer's description, and that it was described most fully and earliest in point of time by Addison; but maintains that Biermer's communications have made important additions to the full understanding of the disease, a claim which need not be disputed, though equal praise is due to Wilks, to Immermann, to Quincke, and to Eichhorst himself.

A lecture by Dr. Stephen Mackenzie, in 1878, gave the most complete account of this disease which had hitherto appeared in England. The credit of its paternity as a distinct disease is admitted as without question due to Addison, and a careful summary is given of its clinical and pathological characters. Of the three cases mentioned, the only one in which a post-mortem examination was made (No. 86 in the table) is remarkable from the early age of the patient. The ophthalmoscopic appearances are figured.

Dr. Magee Finny, in a lecture delivered at the City of Dublin Hospital and published in the first number of the 'British Medical Journal' for 1880, gives a full and interesting account of three cases of idiopathic anæmia, two of which ended in recovery under treatment by arsenic and one died. Unfortunately, from the same cause which spoilt one of Addison's own cases, it was impossible to obtain a post-mortem examination.

Tables of temperature and figures of the blood-corpuscles accompany this lecture, which may profitably be compared with those of Dr. Bramwell's paper and with those at the end of Prof. Eichhorst's monograph.¹

The next important contribution to the subject was in Dr. Coupland's valuable 'Gulstonian Lectures on Anæmia' (1881). The whole of the second lecture is devoted to idiopathic anæmia. The author agrees with Dr. Mackenzie and Dr. Finny as to the question of priority, but does full justice to the observations of Lebert, Biermer, and Immermann. I shall revert to Dr. Coupland's views on the relation of anæmia to chlorosis further on.

RELATION OF ADDISON'S TO OTHER FORMS OF ANÆMIA.

PATHOLOGY OF ANÆMIA IN GENERAL.

What is the result of this accumulated knowledge of nearly thirty years? What more do we know of the nature and causes of anæmia generally, and of this idiopathic and severe form in particular?

There are two ways in which we may study a disease. When the morbid physiological process and its origin have been ascertained, it is, for the purpose of teaching, most desirable to follow the actual sequence of its development; to begin with its necessary antecedents, describe the anatomical changes which it produces, and thence deduce signs by which its existence may be recognised during life. Thus, a surgeon, in describing popliteal aneurism, would begin with an account of the healthy structure of the artery, the mechanical strains to which it is subject, the histological changes which ensue, and the physical results of normal blood-pressure exerted on a diseased vessel. From a consideration of the anatomical connections of the artery would then be deduced the symptoms and diagnostic signs of the tumour. But this was certainly not the course of discovery. Among various swellings of the leg, painful and pulsating tumours were gradually distinguished, and only when their

¹ Compare also the figures of Davy and Mackern ('Lancet,' p. 642, 1877), and Quinke ('Deutsches Arch. f. kl. Med.,' Bd. xx, Tafel i).

origin in a diseased artery was ascertained did investigations follow into the morbid changes of the vessel, and their probable cause.

In the same way, we can now begin an account of scabies by a zoological description of the itch-mite and its habits, and can show how the disease is developed by a series of pathological events which result from the presence of the parasite. But the history of the knowledge of scabies is very different. From the heterogenous cases called *psora* by the Greeks and *scabies* by the Romans, those were gradually separated which were eminently contagious and which affected definite regions, and it was not until long after the disease had been recognised and its cure discovered, that its real origin was demonstrated.

If we apply the same analytical method to the investigation of anæmia, we should begin by collecting all cases of illness in which the surface of the body is obviously pale. In every such case of "want of blood" we should find that the pallor is chiefly due to diminution in number of the red corpuscles of the blood, with frequent deficiency of hæmoglobin in each corpuscle as well. We can recognise as the necessary physiological results of such a condition most of the symptoms which clinical observation shows to accompany anæmia, whatever may be its origin. In proportion to its degree, we find more or less marked breathlessness from lack of the organs which convey oxygen to the tissues, muscular weakness, particularly weakness in the muscular fibres of the heart, coldness of the feet, and passive exudation which takes the form of œdema of the integuments, the meninges and the lungs, with more or less effusion into the serous sacs; diminution or suppression of the menstrual discharge in women, functional murmurs in the heart and blood-vessels, giddiness and faintness, dilatation of the pupils, and torpidity of the muscular coat of the intestines. Lastly, we find more or less pronounced fatty degeneration of the heart and other organs, including the intima of the arteries and capillaries, and, probably as a result of the brittle state of the latter, ecchymoses or more extensive hæmorrhage.

All these symptoms are directly dependent upon anæmia, and accompany it in proportion to its severity and duration.

In classifying this natural group we may first separate those cases of anæmia in which the cause of the want of blood is direct and obvious. Such is the bloodlessness which follows hæmorrhage from a wound or flooding in parturition. The pallor caused by large losses of blood in a healthy subject is accompanied by the muscular weakness, the rapid and irritable pulse, and many of the other signs above noted. But, in most cases the lost corpuscles are quickly reproduced when food and iron are supplied. When, moreover, anæmia is due to direct hæmorrhage, traumatic, puerperal or hæmorrhoidal, in otherwise healthy subjects, certain of the symptoms above mentioned are usually absent.

On the other hand, in cases of primary anæmia we find the symptoms of hæmorrhage most completely and fully developed, and others added beside.

Intermediate between the two extremes may be placed the anæmia of rheumatism, of lead-poisoning, and of phthisis, in which the bloodless state of the patient is secondary to another condition, but is not the result of hæmorrhage.

As in all natural classifications, there is no sharp line to be drawn between the several groups. Even after traumatic hæmorrhages in a healthy subject, recovery is not always rapid and complete. In a case which came under my own observation, a British officer at one of the Chinese ports was waylaid and nearly murdered by assassins. He received several stabs and lost a very large quantity of blood, but recovered from the immediate effects of the wounds and returned to England. He was still, however, when I saw him several years later, extremely anæmic. Similar cases of protracted or even permanent anæmia following puerperal hæmorrhage are not infrequent. Although epistaxis, hæmorrhage from piles, and even hæmaturia in young and healthy men, are commonly recovered from with surprising rapidity, most of us have met with cases in which a single severe hæmorrhage of the kind has been the origin of protracted anæmia with its attendant symptoms.

A second natural group of cases of anæmia is made by those which, without being preceded by hæmorrhage, follow other exhaustive discharges. Severe anæmia may result from frequently recurring pregnancy, from long continued lactation,

from chronic and profuse leucorrhœa, from chronic albuminuria, and from frequently recurrent diarrhœa or chronic dysentery. It also appears in those rare cases of periodical flow of lymph recorded by Lebert and one or two other trustworthy authorities. As with the effects of hæmorrhage, so these forms of anæmia are curable by food and iron.

Next would follow cases in which we have evidence, not of increased loss, but of diminished gain to the blood. Pallor accompanies inanition produced by stricture of the œsophagus, by chronic dyspepsia, by gastritis, and by gastric ulcer even when unattended with hæmorrhage.

Distinguishable from these, at all events in theory, are cases of anæmia which depend upon an increased destruction of red corpuscles, and in which the pallor of the surface is more or less constantly combined with excess of pigment in the urine. The anæmia of acute rheumatism, of phthisis, and of chlorosis, and that which follows typhus and other acute diseases, agree negatively in not being caused by hæmorrhage, nor by profuse discharges, nor by deficiency of food.¹

Another natural group of anæmia is made by those cases in which deficiency of blood, in all its essential constituents, red disks, leucocytes and proteids, appears to depend upon some poisonous effect of drugs. Such are cases of anæmia from mercury, from lead, and from alkalis, and probably allied to these are cases of anæmia from syphilitic and malarial cachexia.

Next may be grouped forms of anæmia which are distinguished by being accompanied by structural changes, by enlargement of the spleen or other lymphatic organs, with or without leucæmia. Compared with the preceding conditions, these may be regarded as not merely frequently recurring

¹ I may here remark what must have frequently struck other observers, though I do not remember to have seen it mentioned, I mean the prominence of anæmia as a symptom of cardiac disease in children. It is comparatively rare to see a child with the dropsy, jaundice, albuminuria, and other familiar symptoms which make up the picture of chronic mitral disease in the adult. When these do occur in children, it is toward the end of the malady, and then the chance of restoration to comparative health by treatment is far less than in the case of adults. The more usual appearance in a young subject of mitral disease is that of a pale, thin, delicate form, closely resembling the familiar aspect of pulmonary phthisis.

morbid states (Symptomen-complexe), but more or less defined "diseases." (Krankheitseinheiten.) I refer to anæmia lymphatica or Hodgkin's disease, and to splenic leuchæmia, which may fairly be called Virchow's disease.

The latter, though recognised later in point of time, has been the more successfully investigated of the two conditions, and is broadly distinguished from all other kinds of anæmia by the diminution of red corpuscles being accompanied by a marked increase of the white, as well as by its association with hypertrophy of the spleen. Closely allied to this are the forms of leuchæmia with enlargement of other organs of analogous structure, especially leuchæmia myelogenica.

Anæmia lymphatica, *i.e.* deficiency of red blood-corpuscles without excess (or without considerable excess) of the white, associated with hypertrophy of lymph-glands, is the disease described by the late Dr. Hodgkin, the history of which has been described by Dr. Wilks in not the least valuable of his contributions to these reports: (Series iii, vol. ii, p. 128; and vol. xi, p. 56.).¹ Many cases are now on record since those first described by Neumann of anæmia combined with similar lymphatic overgrowth in the marrow of the bones, anæmia osteogenica or myelogenica.

Not infrequently a case of leuchæmia splenica is associated with enlargement of the lympharia² or of other cytogenic organs. And there are undoubted cases of true leuchæmia, that is, decided and permanent increase of the leucocytes, occurring without enlargement of the spleen. Such was the case of M. Béhier ('Leucémie intestinale,' Paris, 1868) in which no lesion was present, except hypertrophy of Peyer's patches and the solitary follicles. There are also well-observed cases of leuchæmia myelogenica.

Just as there may be leuchæmia without splenic hypertrophy, so there may be considerable enlargement of the spleen without leuchæmia. And lymphatic overgrowth of

¹ See also Dr. Bright's remarks on the same subject in the 3rd volume of the first series of these 'Reports' (1837), p. 405. Dr. Hodgkin's original paper is in the 17th vol. of the 'Med.-Chir. Transactions' (1832), "On some morbid appearances of the absorbent glands and spleen."

² This word, formed on the analogy of ovarium, seems to be a convenient synonym of lymphatic glands or ganglia (Cf. 'Journ. Anat. and Phys.,' vol. xii, p. 172).

the lympharia or the cytogenic tissue of the liver may be associated with similar isolated nodules in the spleen, without general hypertrophy of the latter organ and without leuchæmia.

There are thus almost innumerable cases which form links between the disease described by Virchow and Bennett and that described by Hodgkin and Wilks. We may name them Leuchæmia splenica, lymphatica, intestinalis, and myelogenica; Leucocytosis (or "pseudo-leuchæmia") lymphatica, thymica, tonsillaris, &c., and Anæmia splenica, lymphatica, myelogenica, &c., according to the condition of the blood and the affection of one or more of the cytogenic organs. Nor when we reach lymphatic anæmia is the chain of gradations ended.

Many cases, no doubt, form a striking and characteristic group in which hypertrophied lymph-glands go with anæmia without increase of leucocytes. These may fairly be named Hodgkin's disease. But other cases are exceptional in the enlarged lymph-glands being the seat of caseous degeneration, so that it is sometimes difficult to draw the line between these and cases of classic "scrofula." Then again we meet with a well-known group of cases, in which only a few glands enlarge, which present the clinical features, not of a general disease, such as might be called anæmia lymphatica, but of a local, usually intra-thoracic, tumour, lymphoma. These in their turn are inseparably allied with cases of malignant disease in which the histological character of the growth leads us to prefer the term sarcoma or lympho-sarcoma to that of lymphoma.

It is only after a survey of these various forms of anæmia which I have attempted briefly to characterise that we can properly appreciate the pathological significance of the idiopathic or essential form of anæmia described by Addison.

It is idiopathic, autochthonous, primary—that is to say, it does not depend on known loss of the constituents of blood, nor on diminished income, nor on increased destruction of formed elements.

There are, however, cases of anæmia which do not follow hæmorrhage, which are not dependent on any primary disease, and which are not accompanied by enlargement of the spleen, the lymph-glands, or any other cytogenic organs, and which

yet differ widely from idiopathic or essential anæmia. They belong to the form of anæmia familiar for centuries as chlorosis or green sickness. It is distinguished not only by its association with amenorrhœa, by the sex and age of the patients, and the prominence of certain symptoms, as constipation and venous murmurs, but also by the readiness with which it is benefited by preparations of steel and by purgatives.

In his Gulstonian Lectures, Dr. Coupland argues against separating chlorosis from idiopathic anæmia. As he seems to anticipate, this would be generally regarded as a retrograde step. It was just because Lebert was content to name his cases of severe anæmia "puerperal chlorosis," and "chloroses particulières," that he failed to recognise a form of anæmia unconnected with menstruation or parturition. Indeed, the chief source of confusion in the study of idiopathic anæmia has arisen from its having been approached from the gynæcological point of view. This particularly applies to the valuable cases published by Professor Gusserow. That the diagnosis between chlorosis and idiopathic anæmia is sometimes difficult I admit. With our present knowledge, it may even, in a given case, be impossible. But, in the absence of characteristic anatomical lesions, every diagnosis by definition of the course and symptoms of a malady is open to the possibility of doubt. All that can be asked is that the groups of concomitant symptoms to which we affix names, and which we use as convenient and brief expressions of facts, should be founded on the natural history of disease, on its origin, course, and event; and that the distinctions we make should prove practically useful. Now, it is not disputed that we meet with cases of profound and ingravescent anæmia, differing from others in their being independent of hæmorrhage and other known causes, in their resistance to ordinary treatment, and in their steadily increasing severity. They are marked by great diminution of red blood disks, without increase of leucocytes, by intercurrent pyrexia, by retinal hæmorrhage, and by symptoms of cardiac weakness. After death such cases show with equal constancy, fatty degeneration of the heart, numerous but small internal hæmorrhages, and slight passive exudations, but no lesion which cannot be ascribed to the primary anæmia. Such cases occur in men, and in women who have

not suffered from amenorrhœa or uterine disorder. It is no doubt possible to argue, as Dr. Coupland does, that cases of anæmia are occasionally met with in lads about puberty which might be called "male chlorosis," that slight rise of temperature may sometimes be observed in chlorotic girls, and that anæmia in young women is not always combined with amenorrhœa. But no one can deny that if we take, not exceptional instances, but the great bulk of cases of "green-sickness," their common characters are widely different from those described by Addison as characteristic of idiopathic anæmia. I do not lay stress on the small heart and narrow aorta observed in cases of chlorosis by Bouillaud and by Virchow, because the constancy of this condition is far from proved, or its absence in other forms of anæmia; nor upon the presence of a normal amount of albumin and globulin in the blood, for this certainly varies independently of the amount of hæmoglobin; nor upon the absence of microcytes, since they have not always been present in cases of idiopathic anæmia. But until we know more of the real cause of anæmia, whether chlorotic or no, it is surely useful to separate cases into groups which correspond with the actual coincidence of symptoms when a large number of instances are taken. For, after all, the object of diagnosis is foreknowledge of results and knowledge of means of relief. In the vast majority of cases which we call chlorosis we can prophecy a favorable termination, and we find satisfactory and apparently direct results from the traditional treatment thoroughly and intelligently carried out. In the cases which agree with the original description of idiopathic anæmia, our prognosis is of the gravest; and we know that, whatever hope we may entertain from other treatment, the ordinary medication of chlorosis and symptomatic anæmia will be useless.

For the present, therefore, it seems in every way desirable to separate chlorosis from idiopathic anæmia in our notions, as they certainly are separated in our practice. Neither one nor the other can be definitely put aside as a completely known "disease," because in both cases we are ignorant of the efficient cause, and in neither are we able to point to any definite anatomical lesion as decisive of our diagnosis.

So far from extending the title of "pernicious anæmia" to

all cases in which, whatever the cause, there is great and progressive diminution of the red corpuscles, I would for the present exclude, not only cases of chlorosis, but those which directly follow menorrhagia or puerperal flooding and those which appear to result from long and severe dyspepsia, chronic diarrhœa, or any other recognised cause of anæmia. In such cases the anæmic condition may be regarded as more or less secondary, and they may properly be grouped with other forms of secondary or symptomatic anæmia. Moreover, in the investigation of a still obscure disease it is, I think, desirable to admit as undoubtedly genuine cases of idiopathic anæmia, those only which have been proved *progressive* by a fatal issue, and “simple” or *essential* by the result of a post-mortem examination.

As a provisional classification of anæmia from a clinical point of view, I would therefore submit the following :

I. A group of cases which agree in being secondary, and symptomatic. Beginning with the simplest kind of anæmia, that which is the direct consequence of traumatic or puerperal hæmorrhage, or of more frequent losses of blood by varicose veins, by epistaxis, by piles, we might next range those cases of anæmia from hæmorrhage which depends on fragility or actual disease of the blood-vessels: hæmophilia, purpura, scurvy, hæmoptysis, hæmatemesis, menorrhagia. Closely allied to these are cases of anæmia which result from losses of pus, lymph, or other discharges, or from the frequent support of a fœtus during pregnancy, or of an infant during lactation. Another group consists of deficiency of blood due to diminished income: the anæmia of starvation, of dyspepsia, of malignant disease of the digestive organs;¹ the secondary anæmia of rheumatism, of fever, of phthisis, of syphilitic and malarial cachexia; and chlorosis, or anæmia with amenorrhœa.

These forms of anæmia all agree not only in being symptomatic, but also in their comparatively slight degree; in the absence of increase of white corpuscles in the blood, or changes

¹ What is called the “cancerous diathesis” need not be considered as a cause of anæmia. Cancer of the breast, for instance, often exists with the aspect of florid health. It only produces anæmia either by hæmorrhage or by its affection of the chylopoietic viscera.

of size or shape in the red; in the absence (at least with very rare exceptions) of pyrexia not due to the primary disease; in the absence of hæmorrhages in internal organs as the result of anæmia; and in amenability to treatment, and especially to treatment by steel.

II. A second group is formed by all cases of anæmia associated with disease of the cytogenic organs, whether or not leuchæmic.

It is important to remember that every case of leuchæmia is also one of anæmia, that excess of leucocytes in the blood is always accompanied by diminution of red disks, and by the pathological and clinical features of lack of blood.

Moreover, leuchæmia and anæmia lymphatica differ from the groups of secondary anæmic states which depend upon increased losses or diminished gain to the economy, as well as from chlorosis, and resemble the idiopathic, primary, or essential form of anæmia described by Addison in the following particulars:—The frequency of hæmorrhages, usually larger in amount in the former, and more numerous in the latter; the very common irregular pyrexia; the severity and ingravescence; the resistance to preparations of steel and more or less marked influence by arsenic; the occasional remittance of symptoms; and the almost invariably fatal termination.

III. The third group of cases are those of which this paper treats:—Idiopathic, primary, or essential anæmia, without any symptoms during life, and without any lesions after death which cannot be explained as directly due to anæmia. The absence of leuchæmia, the almost constant structural changes in the red blood disks, and the absence of notable overgrowth of spleen, lymph-glands, or red marrow, distinguish them from the second group; and their severity and malignancy, the ecchymoses in the retina and elsewhere, the pyrexia, and the almost uniformly fatal result, from the first.

From this point of view I have collected (in Table B at the end of this paper) as many cases as I could find of typical and authentic cases of the disease, having used for the purpose the previous cases published in these 'Reports' by Dr. Wilks (1857) and Dr. Frederick Taylor (1878), as well as those referred to by Lépine, Hermann Müller, and Eichhorst. I

have also made use of the full and conveniently arranged 'Index Catalogue' of the Library of the Surgeon-General's Office issued by the Government of the United States; and have profited by Dr. Neale's useful 'Medical Digest' in referring to cases published in the medical periodicals of this country. The 'Transactions of the Pathological Society' and the post-mortem records of Guy's Hospital since Dr. Taylor's paper was written have also been searched.¹

In the instances where I have not succeeded in seeing the original reports, I have given the authority on which they have been quoted.

I have, for the reasons just given, excluded many of the cases recorded by Liebert, Gusserow, Biermer, Immermann, and Lépine, which have been regarded as examples of essential or pernicious anæmia in women.

I do not pretend to doubt the accuracy of the diagnosis made by these eminent physicians, but for the reasons above given it appears to me instructive to confine our study at present to those cases which conform to the original description of Addison, which are idiopathic, that is, not obviously secondary to known causes of anæmia; simple, that is, not complicated by other conditions; progressive or ingravescent in their course; pernicious or lethal in their result; and which offer not only the characteristic symptoms during life, but also the no less characteristic appearance after death. No doubt every case in which the red corpuscles of the blood are diminished is physiologically connected with every other, from the temporary anæmia of traumatic hæmorrhage to that which accompanies cancer, consumption, or splenic leucæmia. But our best chance of discovering the common condition which connects them all, is to separate them into the most natural groups which pathological and clinical observation can construct.

The admission of some cases will, I fear, seem as arbitrary

¹ While these sheets were passing through the press, a paper appeared in the 'Practitioner' (Jan, 1883), by Dr. J. M. Hobson, in which short notes of a large number of cases were given. Many of these were excluded either as not being primary or not including a *post-mortem* examination, but Dr. Hobson having been good enough to supply me with the references to his cases, I find that all the rest had been already tabulated.

as the exclusion of others. In many, which at first sight seem to be secondary, perusal of the whole history shows that the antecedent fever, or epistaxis, or diarrhœa, or puerperal hæmorrhage was so slight, or occurred so long before symptoms of anæmia, that it cannot be regarded as of causal significance. So, again, old pleuritic adhesions, results of obsolete phthisis, a single cyst in the ovary or the thyroid, are obviously accidental coincidences. Lastly, slight swelling of the spleen, especially after pyrexia, and the pneumonia or other intercurrent disease which sometimes terminates a case, do not in reality complicate it. Thus, for example, Prof. Eichhorst questions the genuine character of a case I reported in Virchow's 'Archiv' because two or three small nodules were found in the spleen, of uncertain origin, but evidently the result of some obsolete process and unconnected with the symptoms. This is the more remarkable because in one of his own cases the pancreas was enlarged ("sehr auffällig gross," p. 163), and in another the mesenteric glands were caseous and there was parenchymatous nephritis (p. 172). So also vague statements of a man of forty-seven, that when young he suffered once or twice from fever, cannot seriously affect the case. Unless our reports of cases are to be intolerably minute, we must decide on the meaning of symptoms and of anatomical lesions, without attempting to justify our conclusions by relating every minute circumstance which formed them, and trust within reasonable limits to the judgment of others—"Hanc veniam petimusque damusque vicissim."

The Pathology of Anæmia.—Of diseases of the blood in general and of anæmia in particular, our knowledge is still very imperfect. For true pathology can only follow anatomy and physiology, and even now Virchow's remark is true "Die Geschichte der rothen Blutkörperchen ist in einem geheimnissvollen Dunkel gehüllt."

No phrases are more common in the mouths of our patients, and even in popular medicine, than "blood-disease," "impurities of the blood," and "poverty of the blood." But we must remember that such terms are the survivals (*superstitiones*) of the long exploded system of Galenical medicine. We do not now believe that blood is one of the four "humours" of the body, or that it is formed in the liver. That a disease is

general or even universal is no proof that it is a disease of the blood. The circulation is often the mere channel for conveying sources of disease, and it is no more reasonable to say that syphilis, or gout, or cancer, is a disease of the blood, than to say the same of poisoning by arsenic or by phosphorus. In fact, it is probable that all changes in quality or quantity of the constituents of the liquor sanguinis are secondary, and that the only affections which can be properly termed diseases of the blood are changes in the numbers or the structure of the red or the colourless corpuscles.

The work of the last twenty years has substantially confirmed the opinion of Hewson¹ that the ultimate origin of the corpuscles of the blood is in the lymph-glands or lympharia. The leucocytes which swarm in these organs are broods of new-born cells, ready to pass into the lymph as lymph-corpuscles and to enter the blood as white blood-corpuscles. There can also be little doubt that the same function is carried on by the other organs, which consist of the same lymphatic, "adenoid," or leuco-cytogenic tissue. The largest, and probably the most important of them, is the spleen. Its Malpighian follicles are precisely the same in structure as the solitary follicles of the intestine and of the tongue and fauces. Similar closed follicles form the Peyerian patches and the tonsils, and make up the substance of the thymus. Diffuse tracts of retiform tissue crowded with leucocytes are found not only in the pulp of the spleen, but under the intestinal and the bronchial mucous membranes, in the interstitial parts of the liver, and in the red marrow of cancellous bone.

These lymphatic structures agree scarcely less in their pathology than in their histology. All of them, thymus, spleen, tonsils, Peyer's patches, and lympharia proper, are at their fullest development and greatest apparent activity in childhood or youth and it is then that they are most liable to disease. All of them become comparatively small as age advances, and shrink, or disappear entirely, at various periods in adult life. The thymus goes first, though it often

¹ "We have proved that vast numbers of central particles [*i.e.* nuclei] made by the thymus and lymphatic glands are poured into the blood-vessels through the thoracic duct, and if we examine the blood attentively we see them floating in it." 'Works,' Gulliver's edition, p. 282.

persists long after puberty, and the spleen last; but the same change affects them all. Enlargement of them is almost always accompanied by anæmia, and the diminution of red corpuscles is accompanied by more or less increase of white; leuchæmia or leucocytosis. Virchow's original view that a hypertrophied spleen manufactures more leucocytes than usual and therefore inundates the blood with them seems probable enough. But why is leuchæmia so rare when the lymph-glands only are enlarged? Why does excess of leucocytes lead to absolute diminution of the red corpuscles in number? and why does enlargement of any lymphatic organs produce anæmia, sometimes with absolute increase, more often with absolute decrease of leucocytes, but always with decrease of red corpuscles? Before we can attempt to answer these questions, we must have better knowledge than at present we possess of the relation between the two kinds of corpuscles and of the mode of origin of the coloured ones.

But the end of the white corpuscles has been almost as difficult to discover as their beginning.

Since the observations of Waller were confirmed and extended by Cohnheim, we have learned the important part which the leucocytes play when they have escaped through a damaged capillary in the process of inflammation. And a steady stream of observation and experiment tends strongly to the belief that in their properties will be found the key to the long-standing puzzle of coagulation after death of the blood.¹ But in the normal condition of health they neither leave the blood-vessel, nor do they help in a process of coagulation. Yet they rapidly diminish in numbers, and it has long been more than a conjecture that they in some way become transformed into red corpuscles. The occurrence of what have been supposed to be transitional forms, in the red marrow and in the blood of the splenic vein, has suggested that the bones and the spleen are the seat of the metamorphosis. Accepting this origin of the red disks as more than probable, it is far from certain by what method they are produced. There is

¹ Recent observations on this subject by Dr. L. C. Wooldridge, who has succeeded in obtaining leucocytes in large quantities from lymph-glands, have thrown important light upon their chemical constitution and their relation to coagulation. ('Proceedings of the Royal Society,' No. 214, 1881.)

no reason to suppose that it is by simple fissure of the leucocytes; this process appears to be always a reproduction of like from like. It takes place in the lympharia, as a method of multiplication of leucocytes; and it takes place in the mesoblast, from which the lymph-glands are differentiated as a method of multiplication of the embryonic blood-corpuscles. But no leucocyte produces a blood disk by simple fission. Nor has recent observation confirmed the ingenious hypothesis, first propounded by Mr. Wharton Jones, that the red corpuscles are modified nuclei. They differ from nuclei in their behaviour to acetic acid, and to staining reagents; and while we have many instances of cells which have lost their nuclei, we have none of nuclei which have lost their surrounding protoplasm—not even the spermatozoa. In the development of red corpuscles in the embryo we see the process of endogenous cell-formation, the brood-cells becoming the disks and the mother-cell the capillary. We can also trace the process of differentiation from the indifferent corpuscles of the mesoblast, like leucocytes or granulation cells, through large, clear, spherical, nucleated cells, to coloured and flattened nucleated disks, like those of the lower vertebrata; and since these contain hæmoglobin throughout their protoplasm, it seems probable that the final small red disk is derived directly from them. Similar red nucleated cells are normally found in red marrow and spleen, and occasionally in the blood of other parts. Thus, for instance, they were observed in the blood of one of the sloths (*Cholopus Hoffmanni*) by the late Dr. Rolleston. Large cells containing red corpuscles in a vacuole have also been seen in the spleen, and have been explained either as brood-capsules with young disks, or as “hæmatoclasts,” which have devoured worn-out old corpuscles, to utilise their hæmoglobin in the production of bilirubin. Recent observations, however, make it probable that though the red disks in the adult are the direct product of the white corpuscles, this is neither by a process of fission, nor by endogenous formation (as observed by Heitzmann in bone) nor by extrusion of a nucleus and assumption of hæmoglobin, as the late Prof. Pouchet, of Rouen, supposed,¹

¹ This view has been supported by a series of interesting observations on the marrow of the guinea-pig and on the blood in embryos of this animal of different

but by a process of budding from the nucleated red corpuscles above mentioned. This, at least, is the hypothesis of Malassez, who has seen such gemmæ, of yellowish-red colour and homogeneous aspect, apparently growing from large nucleated cells, in the spleen and in the red marrow. He dissents from the less probable view held by some Italian histologists that the budding takes place from the nucleus, and has always seen one or more buds attached to the surface of the cell.¹ The process of red disks forming as buds from nucleated and tinted cells, and of indifferent granulation cells becoming large, clear, and yellow, so as to form the nucleated red cells, has also been described as a pathological process by Dr. Creighton,² who applies to these large, nucleated, clear-coloured cells the name hæmatoblast, the term used in a somewhat different sense by Neumann and Bizzozero, and by Hayem.

The absence of nucleated coloured cells or other transitional forms in normal blood seems to show that the development of red corpuscles takes place, not in the blood stream, but in certain organs, and histological evidence points to the red marrow and, less decidedly, to the spleen.

While much remains to be done before a consistent theory of blood-formation can be established, the tendency of observations appears to be to the probability of the following process: that the white corpuscles are formed by fission in the spleen, lympharia, and the other cytogenic organs, that their normal end is to be transformed into large, clear, nucleated cells holding hæmoglobin, and that these hæmatoblasts either extrude their nucleus, or, more probably, give birth by exogenous (or under some conditions endogenous) gemmation to the ordinary small, unnucleated hyaline disks which we call red corpuscles. The seat of this transformation appears to be in the red marrow and in the spleen.

Obscure as the process of blood formation still remains, it is difficult to apply what we know or surmise to the elucidation of anæmia. We must suppose that the colourless cells have

ages, by Prof. Rindfleisch. See 'Archiv f. Mikrosk. Anat.,' Bd. xvii, Tafel iii, figs. 6 and 7. But may not some of these forms be *artifactual*?

¹ See a careful summary of Malassez's observations in the 'London Medical Record' for November, 1882, p. 439.

² 'Journal of Anat. and Phys.,' 1880, xiv, p. 293.

a very transient duration, since though formed in such vast numbers, they yet are comparatively few. As fast as fresh ones are produced, others must be transformed into red corpuscles, unless otherwise disposed of. The astonishing rapidity with which losses of blood are replaced in healthy subjects, also shows that production of red disks is easy and rapid. Even in chlorosis and other forms of non-traumatic anæmia, their multiplication under the influence of iron, measured numerically as well as by the appearance of the patient, is wonderfully rapid; their number is sometimes doubled in a week. If production is thus rapid, it follows from the constancy of their number in health that their life must be short and their destruction equally extensive. Nor are we without means of tracing the destiny of their most abundant and characteristic constituent. For the colouring matter of muscular fibres is proved to be hæmoglobin by its spectrum, and by actually yielding hæmin crystals; and both bilirubin and the yet obscure pigments of the urine and of serum are closely connected with hæmatoidin.

Accepting these physiological data as a working hypothesis for clinical observation, we should expect to find the following pathological states :

(1) Atrophy of spleen or other cytogenic organs associated with diminution of white and red blood-corpuscles (anæmia) and deficiency of pigment in all the tissues and secretions except the choroid and the epidermis.

(2) Hypertrophy of the spleen or lympharia with increase of white blood-cells ("polyleucocythæmia") but no diminution of red disks, of hæmoglobin, or of the derived pigments.

(3) Increase of leucocytes, with diminution of red disks (leuchæmia) and without structural changes, a condition which would point to normal production of the former and destruction of the latter, with deficient activity of the process of transformation of one into the other.

(4) An opposite condition: increased richness in red disks with diminished number of leucocytes, which would indicate increased activity of the same process of transformation. In both cases we should expect to find some changes in the red marrow, and perhaps in the splenic pulp.

(5) Lastly, we should expect a kind of anæmia in which

a diminished number of red disks, with only relative increase of leucocytes, would point to more rapid destruction of the former, and would be associated with more abundant pigmentation of tissues and secretions.

Under the first head may be placed the physiological anæmia of age. The spleen is shrunken, the last vestiges of the thymus have long ago disappeared, the lympharia are small and hard, the agminated follicles of the fauces and the small intestine have atrophied, and only the diffuse cytogenic tissue of the digestive mucous membrane and the red marrow of the bones remain without much diminution. But we have no instance of anæmia from this cause as a pathological condition.

There is no recorded state of increase of leucocytes without decrease of blood disks. In other words, leuchæmia never exists except as a form of anæmia. Nor is the richness of the blood in red disks ever found with diminished numbers of leucocytes. Nor, finally, do we find excess of white corpuscles without hypertrophy of the spleen, or at least of some cytogenic organ.

Therefore mere over-production of healthy leucocytes by hypertrophied and over-active organs, as Virchow describes, is not enough to explain leuchæmia; there must be either a check in their normal development into red disks or else an excessive destruction of the red disks formed.

But excess of leucocytes never occurs without enlargement of one or more of their manufactories. It is, therefore, unlikely that their relative increase is due to want of development into red disks, and probable that it depends upon over-production. This conclusion is strengthened by the observations which go to show that in splenic leuchæmia the leucocytes are large and granular, like those of the spleen, while in lymphatic leuchæmia and leucocytosis they are small and clear with a simple nucleus, like those of the lympharia.

The reason, therefore, for diminution of red blood disks is not impeded development from "hæmatoblasts," but is either actual loss of formed corpuscles by hæmorrhage, or impaired nutrition of the blood as a whole (of red disks, of leucocytes, of hæmoglobin, and of plasma) by excessive loss or diminished supply; or increased destruction of formed red corpuscles, such as may probably take place in syphilis, in malaria, and

in poisoning by mercury or lead. Why hypertrophy of cytogenic organs should apparently always lead to this increased destruction, whether with or without increased formation of white corpuscles, does not as yet appear.

But we have corroborative proof of such destruction in certain cases of anæmia, from the increase of pigment. This seems to be the explanation of the well-known pigmentation of the meninges and many other parts which occurs as the result of ague, a condition recorded by Bright, but fully followed out by Prof. Frerichs. So also the anæmia of syphilitic cachexia is accompanied with excess of pigment. In idiopathic anæmia we have the same evidence of destruction of red blood disks. The urine is often of a deep colour. There is a peculiar yellow tint of the skin which simulates slight jaundice. The fat and the muscles are often of remarkably full and saturated tint, contrasting with the pallid, œdematous look of the other organs; and in one case (Dr. Broadbent's, No. 22 in the Table) the blood gave the hands a yellow stain. Pigmentation of the serous membranes has occasionally been noticed, and it is quite possible that the excess of iron which has been detected in the viscera is derived directly from broken-up hæmoglobin.

On the whole, although we cannot fully explain the pathology of idiopathic anæmia, we may believe that it depends, not upon diminished gains or increased losses to the blood as a whole, nor upon any affection of the cytogenic organs or of the leucocytes, but upon too rapid and extensive destruction of the red blood-corpuscles.

Name.—We have the choice of three titles for this disease, the original one, *Idiopathic anæmia*, given by Addison; *Essential anæmia*, used by Lebert and by many French writers, and *Progressive pernicious anæmia*, the term invented by Biermer and commonly used in Germany.

To the last name there are two objections. The disease is not always progressive in its course; for even in fatal cases temporary improvement has often been observed: and it is not always "pernicious," if, as seems intended, the epithet is to signify malignant, for several patients have recovered when nothing was wanting to complete the diagnosis but their death. "Anæmia gravis" would be a better phrase to

express the extent of bloodlessness, and would correspond to the term *Icterus gravis*. But the true note of this malady is not its severity, nor even its fatality, but its being primary, not the result of hæmorrhage or of organic disease, and simple, not part of a more general condition. The term "essential" expresses this well enough, but since "idiopathic anæmia" was the name earliest given, there can be no reason to use any other until we can replace it by the result of greatly increased knowledge. The term "essential febrile anæmia," suggested by Immermann, is too restricted, since certain cases, alike in all other respects, are apparently apyrexial from first to last. Dr. Pepper proposed the name "anæmatosis," which scarcely seems more distinctive than anæmia.¹ The epithets "simple" or "primary" have been sometimes used instead of "idiopathic," and "fatal" instead of pernicious.

In Table B the title used by each author will be found in the first column.

Geographical distribution.—At least in England, idiopathic anæmia is certainly one of the rarer diseases. In this hospital, where exceptional interest has been naturally taken in it, we have probably had more than an average number of cases sent in, and it is unlikely that any have escaped notice. Yet in nearly thirty years we have not quite thirty cases to record. Judging by published cases in periodicals, as well as by private inquiries, I believe that the disease is not more common in other parts of the United Kingdom. In France, essential anæmia has also been accounted a rare disease, and probably most of the cases have been published. In Germany "pernicious anæmia" is apparently more common, but this partly arises from the more extended signification which has been given to the title employed.

Among the hard working and hard living peasantry of certain parts of Switzerland idiopathic and fatal anæmia would seem to be almost endemic, even if we exclude the large number of recorded cases which either are secondary to

¹ Such terms as "spanæmia," "hypémie," and "oligæmia" have been supposed to express "poorness of blood" more accurately than the word "anæmia." But this is the same pedantry which picks holes in phrases like *delirium tremens*, or *paralysis festinans*. A privativum does not always denote logically complete negation: ἀπρόσωπος, means ill-faced, not literally without a face, and ἀναιμος has exactly the same meaning as the English word bloodless, or the Latin *exsanguis*.

uterine hæmorrhage or malaria, or are otherwise complicated. Lebert, Güsserow, and Biermer all observed their cases while professors at Zürich¹, and have not, I believe, published any more from Silesia or from Alsace. Immermann's cases came from the Canton of Basel and Quincke's from Bern. I have only met with one case reported from Italy which is clearly to be referred to fatal and idiopathic anæmia (No. 17* in Table B). Prof. Rosenstein has met with a single case in Holland, and others have been recorded in Sweden and Denmark. In Spain, in Italy, and the Levant, anæmia is extremely common, but appears to be most frequently due to malarious cachexia; and in the tropics, in addition to this cause, there is in many parts the endemic prevalence of the *Anchylostoma duodenale* in the intestine, or of *Bilharzia hæmatobia* in the pelvic veins. A case of death due to anchylostoma has been published as "progressive pernicious anæmia." A few undoubted cases of idiopathic anæmia have been carefully recorded in the United States and in Canada.

The locality of each case is indicated in the first column of the Tables.

Sex, age, occupation.—Of the cases I have collected 48 occurred in men and 59 in women. But it is obvious that if we were to include all the cases of fatal or severe anæmia which are clearly secondary to uterine hæmorrhage, the proportion would be greatly altered. Thus, of forty-four patients observed at Zürich, only nine were men. Eichhorst's collected figures give sixty-five women to thirty men; but, excluding secondary anæmia, the idiopathic cases are nearly equally divided between the sexes, eleven men to twelve women. In Dr. Coupland's lectures he mentions 110 cases, of which fifty-six occurred in men and fifty-four in women. Dr. Bristowe, in the third edition of his deservedly trusted treatise on medicine, says that "the other form of idiopathic anæmia" (that is, Addison's form as distinct from chlorosis) is chiefly met with in men. So that he would exclude most of the cases which follow parturition or menorrhagia.

Idiopathic anæmia is most frequent in the earlier adult

¹ "Der Kanton Zürich scheint ein auserwählter Herd der Krankheit zu sein." Müller.

years. The average age of the cases I have collected would be thirty-four and four months, but (what is more important) the most frequently occurring ages fall between twenty-one and forty-seven. The youngest age is seven, and the oldest sixty-eight.

The following table shows the results obtained by four collectors :

	II. Müller.		Coupland.		Eichhorst.		Pye-Smith.	
	Male.	Female.	Male.	Female.	Idiopathic.	Secondary.		
Under 15	0	0	1	0	0	1	6	
15—20	2	4	2	4	2	1	4	
21—30	2	9	8	18	4	17	29	
31—40	1	19	9	16	8	22	26	
41—50	2	2	19	9	4	16	21	
51—60	2	1	14	7	4	9	13	
61—70	—	—	3	—	1	1	4	

It must be remembered that many of the cases are common to all four lists.

Idiopathic anæmia, unlike chlorosis and secondary anæmia, is not specially a disease of great cities. Probably it is most frequent among peasants. With some exceptions, as No. 85, and the case related at p. 277, it is met with among the poorer classes and in hospital practice.

Predisposing causes.—The remarkable fact is that we find progressive and fatal anæmia to come on with no assignable cause; and this being admitted, one may well doubt whether preceding dyspepsia, or slight hæmorrhage, or pregnancy, should be regarded as more than a coincidence. If we admit the secondary form of progressive anæmia as “the same disease” with the primary, the following are the most frequent predisposing causes according to other authors.

	M.	F.	
Pregnancy and the puerperal state	—	29	20
Dyspepsia and Diarrhœa	12	12	—
Various discharges	5	7	—
Privation	2	5	11
	Eichhorst	Coupland.	

To these causes Wilks,¹ Lépine, and Coupland add mental shock, grief, or some other nervous influence.

¹ In the ‘British Medical Journal’ for Nov. 28, 1874, p. 680, Dr. Wilks quotes cases which followed grief or fright.

According to Biermer, "der Beginn mit Chlorose ist ungewöhnlich." Ague, also, so frequent a cause of chronic anæmia, seems seldom or never to have been met with as an antecedent of the progressive and fatal form. But one cannot help suspecting that some cases with enlarged spleen and liver and pigmentation may have really been malarious.

SYMPTOMS DURING LIFE. PROGNOSIS.

The more "simple," uncomplicated, and idiopathic the case, the more closely do the symptoms correspond with the original description by Addison.

The approach is gradual and insidious, so that it is often difficult to fix the date of the invasion of the disease. Sudden and definite onset is probably to be explained, as in Prof. Purser's case (No. 45), by a misapprehension of the patient. We constantly meet with the same fallacy in the account given us in private as well as in hospital cases, particularly as to the origin of other chronic diseases, including cases of so-called *phthisis ab hæmoptoë*. The patient tells us that he has been perfectly well, "never had a day's illness," until some slight exposure or supposed injury or strain, to which he attributes all that followed. In fact, few patients fail to provide us with ætiology as well as diagnosis, and the one is usually as trustworthy as the other.

Pallor.—This is always extreme, and extends to the mucous membranes as well as the skin. In the best marked cases it is not the dead whiteness of Bright's disease or phthisis, nor the olivaster tint of chlorosis, nor the earthy hue of malarial, syphilitic, or plumbic cachexia. It is a clear, "waxy" colour; that is, white tinged with yellow, without admixture of carnations, of browns, or of greys. Along with this goes the full yellow colour of the adipose tissue after death, which can sometimes be recognised beforehand in the sub-conjunctival fat, and has been described as a slight degree of jaundice.

Circulation.—The character of the pulse is that of anæmia from any cause. It is rapid and compressible, not small, at least early in the disease, but often jerking, and not unfre-

quently dirotic. Systolic murmurs are usually heard over the cardiac region, sometimes loudest at the base and sometimes at the apex. The former we may perhaps ascribe to the formation of a fluid vein in the ascending aorta, the latter not improbably to regurgitation from defective muscular constriction of the left ostium in systole. However, they are certainly "functional" murmurs. But I know not how to interpret the basic murmur in diastole, which has been noticed in a very few cases and for a short period of time. Whatever its meaning, all observers agree that both mitral and aortic valves are unaffected in this disease, a fact of importance in reference to Dr. Goodhart's cases of valvular disease as the apparent result of anæmia. Arterial murmurs are as constant as cardiac. The characteristic venous hum (bruit de diable, Nonnengeräusch) is also frequent. Dilatation of the heart is rare and hypertrophy still more so, but the weight of the organ appears to be better preserved than in cases of phthisis.

Respiration.—Dyspnoea is sometimes the first symptom complained of. It increases with the diminution of hæmoglobin, but not so rapidly as other symptoms, and is often absent while the patient lies still. It has, at least in one instance, proved the immediate cause of death.

Digestive organs.—In not a few cases vomiting and occasional diarrhoea are marked symptoms, and this when no gastro-intestinal symptoms have preceded the appearance of anæmia. Anorexia and some degree of nausea are almost constantly present.

Edema to a slight extent is seldom wanting, and is occasionally extensive enough to be called anasarca, but I have never seen or read of the characteristic dropsy of eyelids, loins, and genitals, so familiar in cases of Bright's disease. Ascites, hydrothorax, and hydropericardium are often observed but are seldom excessive.

Urine.—The urine in some typical cases of idiopathic anæmia is of a deep clear colour; one of the characters which point to destruction rather than deficient formation of hæmoglobin. Urea has been generally found deficient, falling under twenty and sometimes under ten grammes per diem. This is probably from loss of appetite and diminished nitro-

genous ingesta. But, in some cases its relation to the food taken seems to show relative increase from wasting of tissues. Slight and transient albuminuria has been occasionally noticed. Chlorides were found deficient by Huguenin. The trace of iron normally present in the urine was found increased in quantity by Dr. Emerson Reynolds, and indican by Senator.

Hæmorrhage.—Since Addison wrote, the use of the ophthalmoscope and the clinical thermometer, and the improved microscopic methods which the last thirty years have introduced, have led to three important additions to the description of the disease originally given.

The first is the existence of retinal hæmorrhages. These were first described by Biermer, and have since been detected in the majority of cases. In some instances, however, neither the ophthalmoscope during life, nor careful inspection after death, has revealed their presence, so that they cannot be considered as a constant symptom. Moreover, they are not only frequent in chronic Bright's disease, but have also been observed in cases of anæmia associated with cancer, with malaria, with scurvy, with uterine hæmorrhage, and with hæmatemesis. The points of hæmorrhage are usually small and may be very numerous.

More than a hundred have been counted in one retina. Uhthoff found most ecchymoses in the layer of nerve fibres and the internuclear layer, several in the ganglionic, and fewest in the two nuclear layers.¹ Good figures of the ophthalmoscopic appearances are given by Quincke,² by Finny and by Mackenzie. They are usually clustered around the optic disk. Mantz has described minute varicose dilatations of the capillaries, like those discovered by Charcot in the brain, and Eichhorst figures the same condition. But Nykamp failed to find it, and ascribes the hæmorrhage to diapedesis. In other cases simple rupture has been seen. Atrophic patches are not infrequent, and optic neuritis has been twice observed; also œdema of the disk and retina.

Ecchymoses have been observed in the skin (Cases 25, 29, 51, 54, 57, 64, 83), but this is certainly the exception,

¹ "Die path. Anat. Retinalveränderungen bei progr. Anämie" ('Klin. Monatschrift f. Augenheilkunde,' No. 12, 1880).

² 'Deutsches Archiv,' xx. See also Dr. Gowers' 'Manual of Medical Ophthalmology,' p. 190.

and bleeding from the mucous membranes, hæmoptysis, hæmatemesis, spongy gums, and hæmaturia are rare. The internal hæmorrhages found after death, though often extensive in distribution, are small in amount, and usually punctiform.

Temperature.—Another symptom which was supposed to be pathognomonic of essential anæmia is pyrexia, usually slight in degree and irregularly intermittent in course. The first careful record of the temperature in this disease was made by Prof. Immermann. Since then numerous similar observations have been made. Indeed, more or less febrile action is the rule. Cases, however, have been carefully watched and have ended in death without any rise of temperature being discovered. Moreover, similar febrile symptoms occur in anæmia lymphatica and in leuchæmia.¹ Pyrexia often disappears for days or weeks together.

In the last stage before death, when the disease runs a typical and uncomplicated course, there is often observed a decidedly subnormal temperature. In these cases a cadaveric odour has been noticed for hours or even days before death.

Microscopical characters of the blood.—The first observations were made by Wilks in 1857, when he ascertained that the number of white corpuscles is not materially increased. This has been confirmed by subsequent observers, though it has been stated that a slight and transient leucocytosis has been occasionally seen. In several of the best observed cases the number of the leucocytes was probably diminished even in comparison with that of the red disks, and it is possible that any apparent increase is only relative.

So far as my own observations go, white corpuscles do not show any diminution of amœboid movement on the warm stage, a diminution which has been described by Dr. Cavaſy in leuchæmia, and which was certainly present to my knowledge in one instance of that disease.²

The red corpuscles are not only diminished in number, as tested by the *compte-globule* or one of its modifications, but are also paler from deficiency of hæmoglobin. So that the

¹ Biermer's attempt to explain the pyrexia on "humeral" grounds and Immermann's suggestion of contracted capillaries and increased tissue change are well criticised by Eichhorst.

² In one case Messrs. Davy and Mackern noticed amœboid movements of the large granular leucocytes, with immobility of the smaller ones.

total lack of colour shown by the hæmochromometer is due partly to the fewness of disks, and partly to the paleness of those which remain.

In some cases the diminution has been very remarkable. In one instance, the number was at first 1,100,000 per cubic millimètre—about a fourth of the normal amount¹—and it afterwards fell to less than half of this, namely, 425,000, *i.e.* using Dr. Gowers' nomenclature, the hæmic unit was 8·5. This case recovered, the number gradually rising until it nearly reached five millions. In another case ('Jahresbericht' for 1880, ii, p. 248) the number increased during four weeks from 750,000 to 2,500,000. Lépine noted on the day before a patient's death only 378,750 per cubic millimètre. Worm Müller has seen an equal deficiency in red corpuscles (360,000) repaired, and the patient recover, under the use of arsenic.

In ordinary symptomatic anæmia the number of corpuscles is rarely diminished below one half. In Bright's disease, cancer, phthisis, and even in extreme chlorosis, 1,300,000 (about a fourth) is the lowest number I have seen.

Eichhorst states that the red blood disks are larger than normal, 8 or 9 micromillimètres being the most frequent diameter instead of 7 or 7·5. Kahler, of Prag, finds 9·12 μ the average size, while not unfrequently disks of 12 or even 14 μ in diameter have been seen.

If this is a constant fact, it is another difference between idiopathic anæmia and chlorosis; for in that disease, as in other kinds of secondary anæmia, according to Hayem, the vast majority of the disks are diminished in size, although there may be a small number of "giant" disks of 10 or 12 μ .

That the ordinary blood disks, whether larger than usual or no, are pale, has been observed by most authors. The contrary statements refer, perhaps, to the small spherical corpuscles mentioned below.

The richness of the blood in hæmoglobin is gradually diminished, and may fall as low as a tenth, *i.e.* the blood is as pale as normal blood diluted with nine parts of water. The result is that, apart from its small amount, the blood is, in typical cases, of a pallor which is obvious to the eye, "like infusion of roses," "like muscle washings," or like carmine

¹ Malassez fixes this at 4,000,000 to 4,500,000; Vierordt at 5,000,000.

solution. Whether this pallor is relatively equal to the diminution of blood disks is not clear. In case No. 37, Hayem found that with 500,000 disks per cubic millimètre the colour of the blood was diminished from 100 as the normal to 10, *i.e.* there was no diminution of hæmoglobin in each corpuscle. The red disks in this case were large.

Beside the diminution in number of the red corpuscles, there is in most cases a remarkable variation in their size and shape, which has been described as *microcytosis* and *pœcilocytosis*.¹ The first notice I find of this is by the late Dr. Leared in his case published in the 'Pathological Transactions' for 1858. He writes: "The blood disks presented the peculiarity of being very variable in size, many of them being of an oval shape, while others had the ordinary nummulated appearance."

In 1876 Quinke and Eichhorst published independent accounts of the form and size of the red corpuscles. The latter author described the following conditions as peculiar to "malignant" anæmia. His observations have been since corrected, but in the main confirmed.

(1) Certain disks have been found much smaller and of a much deeper colour than usual. These have been named microcytes. Though very frequent in idiopathic anæmia, they are not absolutely constant, and they may be met with in smaller numbers in normal blood.

(2) The shape of the corpuscles has been found altered. They lose their characteristic discoid figure, no longer form rouleaux, and are crenated, irregularly shrunken, globular, pear-shaped or twisted. Crenation is a phenomenon of no pathological significance. Those who, in teaching histology, have had the opportunity of seeing specimens of blood from a number of healthy young men are well aware of this, and will, I believe, also agree that other changes of shape may occur after blood is drawn which is not in any way morbid.

(3) In a case under Dr. Moxon, in Stephen Ward (No. 39 in the Table), very careful observations of the blood were made by Dr. Davy (now of Exeter) and Dr. George Mackern, then students in the hospital ('Lancet,' September 26th, 1877). The effects of evaporation and change of temperature were

¹ A name invented by Quinke on the analogy of pœcilothermic.

guarded against, and control observations were made on the blood of other patients suffering from phthisis, chlorosis, and Hodgkin's disease. They, like other observers, found that the red disks varied considerably in size and shape and did not form rouleaux; but they also noticed that the hæmoglobin collected together and then oozed out of (or was expelled from) the corpuscles. This separation of the hæmoglobin from the stroma, first observed by Brücke, and somewhat fancifully called exit of the zooid from the œcoid, is well known as the constant result of adding astringents to blood disks, and has also been observed (by Dr. Braxton Hicks and others) as a spontaneous process. Whether it denotes excessive amœboid activity from imperfect differentiation of the protoplasm, or whether it is a natural mode of death and disintegration, this phenomenon cannot be considered constant in idiopathic anæmia. It has, however, been observed and figured by Dr. Bramwell.

(4) Nucleated red corpuscles have been seen in cases of fatal anæmia, generally globular instead of discoid, and larger than ordinary. These occur normally in the red marrow and in the splenic blood. They have also been seen in the circulating blood of mammalia.

(5) Riess has observed the presence of large cells containing red disks within them, "giant cells" such as are found in the spleen and the red marrow.

(6) Max Schultze's granular masses have sometimes been seen in unusual abundance.

There can be no doubt that each of the changes just noted does occur in the blood of idiopathic anæmia. But none of them is constant. They have been looked for by competent observers¹ in undoubted cases, and one or other or all have been absent. Moreover, the presence of nucleated red corpuscles and of microcytes has been noted in cases of medullary anæmia by Cohnheim; indeed, some of the best marked cases of pœcilocytosis, reported as idiopathic or pernicious, may really have been myelogenetic. Microcytes have also been found by Osler and by Laptschinsky, not only in febrile blood, and in that of phthisis and of anæmia lymphatica, but also in that of healthy persons.

¹ *E.g.*, Grainger Stewart, Rosenstein, Lépine, Bradford of Boston.

In two cases of anæmia, one fatal and apparently idiopathic, the other secondary to gall stones and cured, Dr. Samuel West states that the blood was found to contain, in the former, deeply-coloured microcytes and irregular red-disks, and in the latter pear-shaped red corpuscles larger than usual, and disks in which the colouring matter had separated from the general mass of the corpuscle and was collected into a round, strongly refracting globule in one corner. In some cases it had escaped and a pale greyish, slightly granular cell body (œcoid) remained.¹

In the extended observations made by Manassein, it was found that the rapid anæmia produced by venesection in the lower animals was accompanied by some increase in size of the red corpuscles. Hayem found, however, no such changes shortly after hæmorrhage in human beings.²

Diagnosis.—This will depend on the definition which we give of the disease and the rigour with which we apply our definition. The diagnosis can never be considered absolutely certain during the patient's life. But where we find the following characters during life we may, with very great probability, make the diagnosis of "idiopathic anæmia;" that is, we may expect that the issue will be fatal, and that after death nothing but the effects of anæmia will be found. The characters are:—(1) Absence of organic disease, and of any recognised and sufficient cause of anæmia. (2) Absence of uterine and ovarian complications; amenorrhœa, menorrhagia, pregnancy, post-partum hæmorrhage, &c. (3) Severe and ingravescent anæmia. (4) Irregular and occasional pyrexia. (5) Retinal hæmorrhage. (6) Diminution in number of red blood disks without leuchæmia, and presence of small, deeply-coloured red corpuscles. (7) Maintenance of adipose tissue.

Idiopathic anæmia has been diagnosed in cases of Bright's disease and of valvular disease of the heart, of phthisis, of cancer of the stomach, and of other internal tumours.

Occasionally it has been mistaken for morbus Addisonii; but this disease is almost as different in its clinical aspect as in its pathology. In idiopathic anæmia the patient is of

¹ St. Bartholomew's Hospital Reports, vol. xiii, pp. 218, 219.

² See also an interesting communication made by Quincke to the Physiological Society of Berlin on December 29th, 1882.

clear waxen pallor, breathless, and fairly nourished or even fat. In the disease distinctively known as Addison's the patient is only moderately anæmic, thin, bronzed, and despondent; he is not subject to retinal hæmorrhages and the blood is not abnormal. On the other hand, disease of the adrenals is often complicated with phthisis or with caries of the spine. An attempt made by Dr. Pepper to unite the two diseases under the name "anæmatosis" ('*American Journal of the Medical Sciences*,' April, 1877), supported by a comparative table of symptoms (p. 350), only serves to bring out their essential differences. There are, however, at least three cases on record in which the symptoms of anæmia during life have been found after death associated with simple atrophy of the adrenals. The first is one of Biermer's cases (No. 37 in Müller's work, p. 142): the patient was a lad of seventeen, and was said to have a characteristic bronzed skin and pigment patches inside the lips. There was retinal hæmorrhage, and fatty degeneration of the heart was found after death. But the adrenals, though very pale and small (the weight is not given) were apparently healthy, and when examined microscopically by Prof. Eberth (and no better authority could be found) they were reported to be normal. A second somewhat similar case was reported by Dr. Wickham Legg in the '*St. Bartholomew's Reports*,' and a third occurred at this hospital, and was lately brought before the Pathological Society by Dr. Goodhart. It will be found recorded in the '*Pathological Transactions*' for 1882.

There is much closer connection between idiopathic anæmia and the various forms of anæmia lymphatica or pseudo-leuchæmia, both in symptoms and pathology. Indeed, the myelogenetic form of the latter affection resembles idiopathic anæmia so closely, that several observers have not unnaturally believed them to be identical, and others have described pernicious anæmia passing into medullary leuchæmia.¹ The two diseases, however, are not the same. Numerous instances of idiopathic anæmia have occurred in which no affection of the marrow was found, as in that which begins the

¹ A case of Prof. Frerichs', described by Dr. Litten in the '*Berliner klin. Wochenschrift*' for May 7, 1877. Compare two cases of osteo-myelitis recorded by Grawitz in '*Virchow's Archiv*' (lxxxi, p. 353), and a paper by Ehrlich in the *Charité-Annalen*.

present paper, and in several others noted in the table at its close. Where there are tenderness and swelling of the sternum, tibia, or other bones during life, or where *post mortem* the yellow marrow (or modified adipose tissue) of the long bones is found transformed into red marrow (or cytogenic tissue), the diagnosis is clear enough. But, at least during life, it must often be obscure.

Course, duration, and prognosis.—Idiopathic anæmia is not a steadily “progressive” disease. Not only is the pyrexia intermittent, but the other symptoms often subside, colour returns, and the diagnosis begins to be doubted. This improvement may be of considerable length, for weeks or months, and may be repeated once or even twice in the course of a single case. But almost always the anæmic condition returns without any assignable reason, even when its first appearance has been attributed to a definite cause.

Occasionally, however, there can be no reasonable doubt that complete recovery has ensued. Addison himself avoided such epithets as “fatal,” “malignant,” or “pernicious” anæmia. He says that “the disease *with scarcely a single exception* was followed after a variable period by the same fatal result,” and again: “with *perhaps a single exception*, the disease, in my own experience, resisted all remedial efforts and sooner or later terminated fatally.”

The following may serve as an example of recovery from progressive and apparently idiopathic anæmia:

CASE.—A patient of Dr. Thurston, of Ashford, consulted me in March, 1880, suffering from marked anæmia. He was fifty-three years old, a stout and well-built man, married, and living in comfort. He had an attack of pneumonia when fifteen years old, and has for many years been subject to ordinary chronic and intermittent psoriasis. He has never suffered from ague, gout, rheumatism, fever, or hæmorrhage. For about six months past he has been imperceptibly losing colour, strength, and appetite. He is now exsanguine, with a sallow tint, the tongue and gums and conjunctiva, as well as the skin, presenting the slightly icteric hue of mitral disease. There is no loss of flesh. He has a short, dry cough, without expectoration. The pulse is of fair volume and strength, but is markedly compressible. There are no vascular or cardiac

murmurs, and the lungs are sound. Temperature normal. The lymph-glands are not swollen, and the liver, spleen, and testes appear to be normal. The urine is dark, free from albumin, but with a doubtful trace of bile pigment. The *fundus oculi* is anæmic, but no hæmorrhage is visible.

The diagnosis then entered was "Either idiopathic anæmia or some obscure internal carcinoma."

After a month I saw him again, and made the following notes:—"Has occasionally vomited, but this Dr. Thurston has succeeded in stopping. On the whole feels better, though still very anæmic and short breathed." No bruit, but first cardiac sound prolonged at apex.

A fortnight later I heard that under steel and strychnia he had greatly improved. In October, however, he called on me again, saying that for some weeks he had been going back and was now as ill as when I first saw him. Anæmia was deeper than before; urine full coloured; œdema of ankles; dyspnœa; no pain, and no other sign of disease. Soon afterwards pyrexia appeared. In February following (1881), while taking arsenic, he was attacked with common eczema of the face, which rapidly spread over almost the whole body. Under these circumstances I saw him for a fourth time, and then detected hæmorrhage in the upper part of one retina, considerable enough to produce limitation of the field of vision downwards when using that eye only. In April I heard that he was no better, but was persevering with Fowler's solution. The eczema gradually disappeared, and in February, 1882, he was well of all his symptoms. I have not seen him since, but Dr. Thurston kindly informs me that the patient continues well, "better than for years past."¹

Other cases of recovery.—Admitting the fallacies of diagnosis to which the most experienced observers are liable in the absence of anatomical evidence of disease, I have selected cases which appear well entitled to be regarded as cases of idiopathic and severe anæmia, in which the general symptoms were confirmed by retinal hæmorrhage, or by a microscopical

¹ While this paper is in the press, another patient, a near relative of Mr. E—, tells me that he continues perfectly well and of good colour, though subject now and then to slight return of eczema. He has never fully recovered the use of one eye.

examination of the blood, and which have ended in recovery. (Table C.)

Of sixty-five cases of "pernicious anæmia," widely interpreted, under Biermer and Huguenin, at Zürich, only four permanently recovered. In twenty-seven cases of primary "pernicious" anæmia collected by Eichhorst, there were only two recoveries; in ninety-four cases secondary to some other condition, only six.

The *duration* of the fatal cases is often difficult to state, from the insidious manner in which they begin. Among seventy-five cases, the shortest time recorded is only seven or eight weeks in eight; it was two to six months in thirty-seven; six to twelve months in twenty; and above a year in ten.

In two or three cases, the patient appears to have suffered from a previous attack of the same disease.

MORBID ANATOMY.

The appearances after death in genuine cases of idiopathic anæmia are strictly consequences of the condition of the blood. The pale, exsanguine condition of the internal organs is no less striking than that of the skin, the gums, and the conjunctiva during life. The heart, the great vessels of the chest, and the portal veins, contain but very little blood; what there is, is pale in colour, and the clots which it forms are thin and scanty. Occasionally, however, enough blood can be collected to fill a narrow glass, and then, so slow is the clotting, that there is time for the few corpuscles to sink, and leave the liquor sanguinis still paler than before. It seems probable that the albumen and globulins diminish in nearly the same degree as the corpuscles.

Some amount of *œdema* is almost always noticed, from a watery infiltration of the meninges and the lungs to general anasarca, with some amount of passive effusion into the serous cavities. The exuded serum is usually of a deep yellow colour, as if it contained more pigment than usual. Hydrothorax of one or the other side is occasionally seen, and more rarely

considerable ascites, or hydropericardium. The presence of shreds of lymph sometimes shows that there has been inflammation as well as dropsy of the pleura.

Petechiæ, whether observed or not during life, can almost constantly be recognised after death, but there is a remarkable absence of cadaveric blood stains (hypostasis).

The body is exceptionally found wasted, even to emaciation, but in the most typical cases subsequent experience has confirmed Addison's description of the remarkable persistence of fat, both under the skin and under the serous membranes. The appendices epiploicæ are especially noted as well preserved. The full yellow tint of the adipose tissue is also remarkable, and the voluntary muscles, instead of being pale and flabby, as in phthisis, are tolerably firm and most frequently of a deep red colour. There are, however, exceptions to this, as in one of my own and in Eichhorst's cases. Fatty degeneration of the diaphragm, abdominal and other muscles, has been noted in several cases.

A remarkable dark grey staining has been repeatedly noticed in the peritoneum, pancreas, spleen, liver, or kidneys. It appears to be due to a sulphide of iron, and careful comparative analyses by Quincke, Grohe, Rosenstein, and Tranchimont, have shown that along with the diminution of hæmoglobin in the blood, the amount of iron in the tissues may be increased. This may, however, be due to the preparations of steel which are often given as medicine.

Addison observed the remarkable interstitial *fatty degeneration of the heart* in the form of zigzag lines across the bundles of fibres—the “tabby mottling” of Dr. Quain. It affects the ventricles more than the auricles, the left ventricle more than the right, and the papillary muscles more than any other part. This condition was fully described by Wilks in 1857,¹ and by Ponfick in 1873,² and is the most constant anatomical condition in the disease. In fact, its absence throws doubt on the genuine character of a case. The cardiac muscle is usually pale and flabby, the cavities occasionally dilated or somewhat hypertrophied, the valves normal. Simi-

¹ ‘Guy’s Hospital Reports,’ iii, 3, p. 218.

² “Anämische Form des Fettherzens” (‘Berliner klin. Wochenschrift,’ No. 1, for 1873).

lar interstitial fatty degeneration has been observed by Wilks, and later writers, in the liver, and less frequently in the kidneys. There is no arcus senilis of the cornea.

Spots of superficial fatty degeneration may be seen (as Virchow observed in chlorosis) in the intima of the aorta and other arteries, and the same change is also stated to occur in the capillary vessels.

That these lesions, and especially the fatty heart, are the result of want of blood is proved from their occurrence in other conditions of long-continued anæmia, and from their artificial production in animals by repeated venesection.¹ The degeneration is probably connected with deficient supply of oxygen.

It has been suggested that degeneration of the capillary walls is the cause of their rupture and the consequent ecchymosis. Although observers have frequently failed to find fatty degeneration of the capillaries of the pia and brain, it is almost certain that deficiency of the constituents of healthy blood acts directly on the nutrition of the capillaries which contain it. The observations of Nykamp² disprove the existence of minute aneurisms as a constant condition, although varicose dilatations certainly occur, as figured by Eichhorst.³

This *hæmorrhage* is as constant, or almost as constant, a post-mortem occurrence as fatty degeneration. It is sometimes extensively distributed, as much as in purpura or malignant fevers, in typhus, or variola, or pyæmia, but it is rarely large. The hæmorrhagic spots in the retina are less in size and far more numerous than in cases of chronic Bright's disease. Hæmaturia, melæna, hæmoptysis, and hæmatemesis are seldom seen, and even epistaxis is not common.

Next to the retina, the most frequent seats of hæmorrhage seem to be the corona radiata, the surface of the cerebrum, and the inner surface of the dura mater (the so-called parietal arachnoid), where slight local inflammation sometimes follows; then the serous membranes and endocardium, the mucous membrane of the stomach, and the lungs. The skin is very seldom so affected. Similar ecchymoses have been

¹ See the experiments by Perl recorded in 'Virchow's Archiv' (1874).

² See his figure in the 'Berliner kl. Woch.,' for Feb. 26, 1877, taken from the post-mortem examination on Prof. Rosenstein's case (No. 41 in Table B).

³ Loc. cit., p. 273.

seen in the œsophagus, the larynx, and trachea, the bladder, the pancreas, and the gall-bladder, also in the interstitial tissue of the muscles and of the orbit.

The *liver*, beside, as above mentioned, being frequently fatty, is often as anæmic as the other viscera, but occasionally retains enough blood to preserve a normal or nearly normal appearance.

In one case Huguenin found the glandular epithelium of the *pancreas* in a condition of fatty degeneration.

The mucous membrane of the *stomach*, beside hæmorrhagic erosion, is frequently the seat of the fatty degeneration of the tubular glands, described by Habershon and by Fenwick in this country, and by Ponfick in Germany. Occasionally there has been observed infiltration of leucocytes between the tubules (a muco-gastritis), and sometimes a thickening of the mucosa, with atrophy of the tubules, which has been described as cirrhosis of the stomach, and may probably be a later stage of the same interstitial process. (Cases 46 and 95.)

The *intestines* are pale and œdematous, with occasional petechiæ or hæmorrhagic erosions.

The *spleen* is less absolutely blanched than the rest of the body. It is sometimes found slightly enlarged, sometimes shrunk. The size will depend partly on the age of the patient, partly on the degree of pyrexia before death. Any considerable enlargement (say to 15 or 18 oz., or above 500 grammes) would suggest the diagnosis of anæmia splenica (pseudo-leuchæmia).

The limit in length of the healthy spleen in growing adults is given in the last edition of 'Quain's Anatomy' (by Prof. Thane), as 5 or 5½ inches, by Henle as 14 centimètres (nearly 6 inches), by Cruveilhier as above an average of 12 centimètres. In three cases of idiopathic anæmia Eichhorst found the spleen 12, 15, and 20 centimètres long. In ten other cases I find the numbers 11, 12·5, 11, 12·5, 12, 15, 17·5, 13, 13·5, and 15 respectively.

A better criterion is the weight of the organ, which is given as varying in health from 6 to 15 oz. by Sömmering, 7½—10½ by Krause, 6—10 by Reid, 4—10 by Ellis, 5—7 by Turner. In twenty-six cases of well-attested idiopathic anæmia I find the following reports of the weight of the spleen :

11 oz. (311 grammes).	"Slightly swollen."
"Somewhat large."	"Small."
"Normal."	300 grammes (about 10½ oz.)
11 oz., "normal."	"Scarcely enlarged."
"Enlarged."	"Enlarged."
"Shrunken."	"Healthy."
200 grammes (7 oz.).	12 oz., or about 340 grammes.
"Normal."	8½ oz. (241 grammes).
"Somewhat enlarged."	7 oz. (nearly 200 grammes).
"Slightly enlarged."	7 oz.
"Somewhat enlarged."	8 oz. (227 grammes).
"Slightly swollen."	8 oz.
16 oz. (453·5 grammes).	10 oz. (283 grammes).

The spleen of simple anæmia differs from the firm, fleshy hypertrophy of leuchæmia, the fibroid enlargement of Hodgkin's disease, the brittle, congested ague-cake, and the soft, swollen spleen of enteric fever.

In genuine cases of idiopathic anæmia the *lymph-glands* are not enlarged, nor are the solitary or agminated lymph follicles. Slight swelling of the mesenteric lympharia has been occasionally noticed, due perhaps to precedent diarrhœa.

The *marrow* of the bones has been found perfectly normal in the majority of cases in which it has been examined by Quincke, Lépine, Bradbury, Burger, and many others. The cases reported by Cohnheim,¹ Scheby-Buch, Pepper, and Gardner and Osler,² where the conditions of anæmia (or pseudo-leuchæmia) myelogenetica were found in the bones, must, I think, be separated from those of idiopathic anæmia, although the symptoms of the two diseases during life so closely resemble one another. It must not be forgotten that the same condition is stated by Neumann to have occurred in one case of Addison's disease of the adrenals.

Nervous system.—The brain has been found excessively anæmic, the pia mater œdematous, and both full of minute ecchymoses. Hæmorrhagic pachymeningitis has also been observed. In one of my own cases the brain weighed fifty-seven ounces (1610 grammes), a high figure but well within normal variations of size, and it was a perfectly healthy brain. In a case of profound anæmia, complicated by valvular

¹ Virchow's 'Archiv,' lxxviii (1876).

² 'Canada Med. and Surg. Journal,' March, 1877.

disease of the heart, and therefore not included in my list, Schumann describes increase in the nuclei of the cerebral neuroglia. In another, Eichhorst found dilatation of the central canal of the cord.

The semilunar ganglia were examined by the accomplished histologist Quekett in one of Addison's original cases, and found to be in a state of fatty degeneration. In a case of "progressive pernicious anæmia," recorded by Dr. Brigidi ('*Lo Sperimentale*,' May, 1878¹), the solar plexus is described as much affected. The changes were the same often found in other diseases: proliferation of nuclei, increase of the interstitial tissue in quantity and firmness, and granular pigmentation of the ganglion corpuscles. Similar conditions have been observed, frequently but not constantly, in *Morbus Addisonii*. But, in the first place, careful examination of the semilunar ganglia and surrounding plexus has in numerous cases of idiopathic anæmia shown that they are perfectly normal.² Secondly, the same condition of atrophy and "cirrhosis" has been found under the most varied conditions.

In a paper just published in the '*British Medical Journal*,' January 13th, 1883, Dr. Saundby criticises the statements made by Da Costa and Longstreth as to its occurrence in chronic Bright's disease, and quotes from several Italian authors' accounts of the same appearance being observed in divers diseases. We have no reason for expecting changes in the solar plexus. The functions of peripheral ganglia are little known and are probably very circumscribed. There is not the least evidence for supposing that they have any relation to blood-making or to pigmentation. Lastly, observation of them in men and animals (*e.g.* of the superior cervical ganglion in rabbits) proves that the amount of interstitial tissue and degree of pigmentation vary within wide limits in healthy subjects.

¹ Quoted in the '*London Medical Record*,' 1878, p. 430.

² "Wilks hat die Semilunar-ganglion mehrmals durchmustert, und sie stets gesund gefunden: dasselbe gibt Pye-Smith an, nur einmal bin ich auf die Bemerkung gestossen dass in ihnen Verfettungen wahrnehmbar waren" (Eichhorst).

TREATMENT.

Most physicians have learned to distrust iron, in whatever doses and under whatever form, as a remedy in idiopathic anæmia. In fact, this is one of the differences between it and chlorosis. Wine, animal food, quinine, and the other so-called tonics have generally proved equally useless.

Phosphorus has been recommended for this as for some other cachectic states of uncertain origin. Like other physicians, I have given it, as I believe, without prejudice, and like others, have not seen advantage from its administration. In one case, in which it was fairly tried for several weeks and in increasing doses, it was thought by the patient to do harm, but this was probably not the case.

Another powerful drug, arsenic, has been used, both in this country and in Germany, in various forms of obstinate anæmia, especially in Hodgkin's disease. In these last cases its administration internally has been sometimes (and apparently with occasional benefit) combined with injection of an arsenical solution into the enlarged lymph glands.

But arsenic is not even mentioned in the monographs of Müller and Eichhorst as a remedy in pernicious anæmia. The merit of using it to good effect in this intractable disease is due to Dr. Byrom Bramwell, who published eight cases in the 'Edinburgh Medical Journal' for 1877 (See Table C, Nos. 11—14). In three of these arsenic was given and recovery followed. Of the rest, arsenic was only given to one patient and he left the hospital shortly afterwards. Dr. Bramwell adds, however, with candour and judgment: "I would not for a moment have it supposed that arsenic will be found a specific for the disease." In three cases treated with arsenic by Dr. Finny, two recovered and one died ('British Medical Journal,' January 3rd and 10th, 1880). In a case reported by Dr. Haddon ('Edinburgh Medical Journal,' xxiv, 493) in 1878, arsenic was given with apparent benefit, but the patient died after leaving hospital, and no post-mortem examination was made.

In my own case, detailed at the beginning of this paper, the effect of arsenic was striking and apparently satisfactory.

By the patient's own testimony, out of the hospital as well as in the wards, and not less by the observation of others, his symptoms were very decidedly improved under its use; it appeared nevertheless to lose its power when repeated, and did not prevent (though I hope it may have delayed) the fatal result.

The temporary improvement which is one of the not unfrequent features of the disease, might make us distrustful of the effect of any drug; and some of the best established cases of severe idiopathic anæmia have recovered without arsenic, or steel, or phosphorus, or any other treatment.

In my case, No. 100, as in others, transfusion of blood was practised. Skilfully performed, there is little danger connected with this operation. There is usually a moderate pyrexia which speedily subsides. For the time the patient's appearance and symptoms usually improve, but it is very doubtful whether in these cases transfusion has ever done more than prolong life.

It proved unsuccessful in the hands of Gusserow, Bradbury, and Bramwell. In one of Quinke's cases recovery ensued after transfusion into the radial artery of 80 c.c. of human blood. He recommends this method; and it would seem to be preferable from avoidance of the risk of air entering the veins and of embolism in the great vessels. But Billroth speaks unfavourably of arterial transfusion. Though transfusion of blood was one of the first fruits of the experiments of the Royal Society in the seventeenth century, we still need the help of direct and extended trials of various methods upon animals.

In four cases recovery followed transfusion, beside the one noticed above. In one somewhat doubtful case of anæmia in a man of fifty-six,¹ the improvement seemed to result from giving up treatment and returning to a country life. The same result followed a removal to the hilly country of Silesia in the case of a patient with apparently idiopathic and very severe anæmia under Professor Frerichs. (Table C, No. 15.)

¹ "Rentier, lebt in den besten Verhältnissen: trinkt nach dem Gebrauch seines Wohnorts, täglich einige Flaschen ziemlich sauren Weins." ('Deutsches Archiv f. klin. Med.,' Bd. xx, S. 15.)

BIBLIOGRAPHY.

(See also the references in the Tables.)

COMBE, 1823. Transactions of the Medico-Chirurgical Society of Edinburgh. (A genuine case with autopsy.)

*PIORRY, 1840. "Traité des altérations du sang." (A single case, probably genuine.) Quoted by Lépine.

MARSHALL HALL, 1838-44. "Principles of the Theory and Practice of Medicine." (A case in a young woman.)

BARCLAY, 1851. Medical Times. (Two genuine cases with autopsy.)

*LAUTH, 1852, and Chalot, 1858. "De la Cachexie séreuse des femmes enceintes et des accouchées." Thèse de Strasbourg.

LEBERT, 1853-4. Report of five cases of puerperal chlorosis in Zürich Hospital, quoted in his "Handbuch der allg. Path. und Ther.," 1876, p. 73.

ADDISON, 1855. Description of the disease in the introduction to his Monograph on "The Constitutional and Local Effects of Disease of the Suprarenal Capsules." Reprinted in his works by the New Sydenham Society, 1868.

WILKS, 1857. Guy's Hosp. Reports. (Seven cases with autopsies and reference to Addison's discovery.)

WILKS, 1859. "Lectures on Pathological Anatomy." 1st ed., p. 459.

*CAZENAVE, 1860. Journal de Médecine de Bordeaux. "Trois observations d'anémie essentielle." (An imperfectly described but probably genuine case in a young man. Quoted by Scheby Buch and also by Lépine, apparently from the same abstract.)

* I have not been able to see the publications marked with an asterisk.

POTAIN. Article "Anémie" in the Dictionnaire Encyclopédique des Sciences Médicales, tom. iv, p. 327. (Though the preceding case of Cazenave and one of Habershon are quoted, there is no mention of Addison's and Wilks's observations, nor any hint of the existence of the disease in question.)

E. WAGNER, 1864. "Die Fettmetamorphose des Herzfleisches in Beziehung zu deren ursächlichen Krankheiten." (A monograph of 159 pages, without drawings. Among the many causes of fatty degeneration, primary anæmia does not appear. But the last of the 144 cases given as primary degeneration with anæmia may probably have been one of Addison's anæmia. No. 14 in Table B.)

*BIERMER, 1868. First communication at the annual meeting of the 'Versammlung deutscher Naturforscher und Aerzte.'

*PERROUD, 1869. Cases of fatty degeneration with anæmia and dropsy; not all of the same kind. One apparently a case of latent enterica. In all more stress is laid on the anatomical secondary changes than on the anæmia.

*CORAZZA, 1869. "Storia di un caso di oligæmia, con riflessioni su quest' affezione, sulla chlorosi e sulla degenerazione grassosa degli organi." (One case quoted from Schmidt's Jahrbuch by Scheby Buch, and from him by Lépine.)

*VAULAIN et MASIUS. "De la Microcythémie." (Brussels, 1871.)

*DOMINGUEZ. "Anémie idiopathique," 1871.

GUSSEROW, 1871. "Ueber hochgradigste Anämie Schwangerer." Arch. f. Gynäkologie, ii, p. 218.

BIERMER, 1872. "Progressive perniciöse anämie." Correspondenzblatt f. Schweiz. Aerzte, ii, No. 1. (A full account of what he supposed to be a new mode of death after pregnancy and parturition.)

PEPPER. American Journal of the Medical Sciences, Oct., 1872. (Two cases.)

PERL, 1873-4. The production of fatty degeneration of the heart in dogs by venæsection. Virchow's Archiv, vol. 59.

PHILLIPS, 1873. "On sudden death from syncope soon after labour." Guy's Hosp. Rep., xviii, p. 159.

*PONFICK, 1873. "Ueber Fettherz." Berliner klinische Wochenschrift, No. 1.

*GFÖRER, 1874. "Tod einer Schwangeren in Folge hochgra-

digster Anämie ohne Blutverlust." *Memorabilien*, vol. xix, p. 116. (No autopsy.)

IMMERMANN, 1874. "Ueber progressive perniciöse Anämie." *Deutsches Archiv f. kl. Med.*, vol. xiii, p. 209.

ZENKER. In the same volume (p. 348) is a case of pernicious anæmia complicated with valvular disease of the heart and other lesions; quite inadmissible.

*SÖRENSEN, 1874. A case of excessive "oligocythemia" (apparently true idiopathic anæmia) with autopsy. Only seen in abstract.

PEPPER, 1875. "Progr. pern. anæmia or anæmatosis." *American Journ. Med. Sc.*, cxi, p. 313.

MANTZ, 1875. Changes in the retina in anæmia. *Centralblatt für die Med. Wissenschaften*, xiii, 675.

SCHÜLE, 1875. "Beiträge zur Kenntniss perniciöser Anämieen." *Allg. Zeitschrift f. Psychiatric*, xxxii, p. 1. (Three cases of extreme anæmia in the course of cerebral disease with dementia. There was, however, also emaciation; and the autopsy showed in each case, beside the results of anæmia, organic disease of aortic valves, brain or cord.)

*FEDE, 1875. *Centralblatt der Med. Wissenschaften*: quoted from 'Movimento med-chir.' tom. vii. (A case of secondary anæmia in the course of malignant osteo-sarcoma.)

PYE-SMITH, 1875. *Virchow's Archiv*. (Two cases, with quotation of Addison's description.)

SCHEBY BUCH, 1876. *Deutsches Archiv*, vol. xvii, p. 467. Historical account. (He quotes Dr. Barclay's two cases and Marshall Hall's and Lebert's, but does not allude to Addison or to Wilks, though he quotes a paper which reproduced Addison's account.)

COHNHEIM, 1876. Affection of the marrow in anæmia. *Virchow's Archiv*, vol. 68, p. 291.

QUINCKE, 1876. Papers in Volkmann's *Sammlung*, and also in *Deutsches Archiv* for 1877 (vol. xx). The latter with ophthalmoscopic coloured drawing of retinal hæmorrhage and figures of blood-corpuscles.

EICHHORST. Notes on the blood in anæmia. (*Centralblatt*, June 24th, 1876).

*ZOLLER, 1876. "De l'anémie pernicieuse progressive." *Thèse*.

MOSLER, Dec., 1876. *Berliner klin. Wochenschrift*. Nos. 49—52. Papers on leuchæmia myelogenetica. (He failed to find nucleated red corpuscles in the blood.)

NEUMANN, 1877. The condition of the marrow in progressive pernicious anæmia. *Berlin. kl. Wochenschr.*, xiv, 685.

ANDREW. *Medical Times and Gazette*, 1877, p. 471. (A discussion of the claim of pernicious anæmia to be accounted a distinct disease.)

LÉPINE. *Revue Mensuelle*, Jan., 1877. "Sur les Anémies progressives." (An excellent historical and critical review with cases.)

HERMANN MÜLLER, 1877. Treatise of 250 pages dedicated to Prof. Biermer. (All the sixty-two cases described occurred in the Zürich Hospital under Biermer or Huguenin.)

GARDNER and OSLER, 1877. Reprint from *Canada Medical Journal*: "Case of progressive pernicious anæmia. (Idiopathic of Addison)." A thoroughly described case, in which, however, there was the affection of the marrow characteristic of anæmia myelogenetica.

*RUKLIN, 1877. "Étude critique sur l'anémie dite pernicieuse progressive." Thèse.

*ROBERT, 1877. "De l'anémie essentielle grave et progressive." Thèse de Montpellier.

BRAMWELL, 1877. Cases in the *Edinburgh Medical Journal* treated by arsenic, with observations on the history of the disease.

PYE-SMITH. Review of Lépine's article and other authorities with quotations of Addison's and Wilks' description in the *London Medical Record* for Aug. 15 and Nov. 15, 1877.

NYKAMP. *Histological Account of the Retina of Idiopathic Anæmia in the Berl. kl. Wochenschr.* for February 26th, 1877.

*REICH, 1878. "Ueber perniciöse progressive Anämie." Inaugural dissertation. Bonn.

THISQUEN, 1878. On the same subject: also an inaugural dissertation for the degree at Bonn.

EICHHORST, 1878. Treatise of 375 pages with drawings of blood and charts of temperature, and a collection of previously recorded cases in abstract as well as some additional cases observed in Prof. Frerichs' wards at Berlin.

TAYLOR, F. These Reports for 1878. A record of all the cases which occurred in Guy's Hospital since Addison's death.

S. MACKENZIE, 1878. Reprint of clinical lecture in the *Lancet* with ophthalmoscopic drawings.

MAGEE FINNY, 1880. A clinical lecture on the disease in Dublin. *British Medical Journal*, January.

UHTHOFF, 1880. Account of the Retina in Idiopathic Anæmia, in the *Monatschrift für Augenheilkunde*, No. 12.

COUPLAND, 1881. *Gulstonian Lectures on Anæmia*, delivered before the Royal College of Physicians. Reprinted from the *Lancet*.

TABLE A.—Cases of Fatal and probably Idiopathic Anæmia, recorded before Addison's description of the disease in 1855.

No.	Author, reference, and date.	Sex and age.	Symptoms during life	Duration.	Post-mortem results.
1	J. S. Combe, 1823 ('Trans. Med.-Chir. Soc. of Edinburgh', p. 194), "History of a Case of Anæmia"	M., 47	Ingravescent and profound anæmia, with no other signs of disease; slight anasarca	9 months	The whole body remarkably bloodless; heart pale and sodden; œdema; hydrothorax; ossification of dura; no other lesion found.
2	? ? Andral, 1823, 'Clinique Médicale, iii, p. 535 "Hydropisie Essentielle"	F., 24	After delivery, anasarca and ascites, with great anæmia supervened	?	All the organs were found bloodless, but apparently healthy. (Edema; hydrothorax.
3	Marshall Hall, 1837 ('Principles of Medicine,' p. 207)	F., 18	After July, 1833, became sallow and pale, with headache and dyspnœa. In 1834 œdema of ankles; slight delirium; asthenia	18 months	Anæmia; no wasting; œdema of pia ("opaque lymph"); œdema of lungs; no phthisis; simple cyst in one ovary.
4	? Piorry, 1840 ('Traité des altérations du sang'), "Polyanémie"	M., 50	Anorexia and depression after venereal excesses; ingravescant anæmia, with syncope, palpitations, &c.	Not stated	Extreme bloodlessness of all the organs; no organic lesion discovered.
5	? ? Pearce, 1845 ('Lancet,' i, p. 643)	F., 19	Habitual anæmia; scanty menses; rapid and extreme pallor, with syncope, palpitation, and cardiac murmur; violent hysteria; low temperature	About a week	No autopsy.
6	Barclay, 1851 ('Med. Times,' May 3, p. 480), "Death from Anæmia"	M., 34	Pain in loins; dry cough; anæmia; dyspnœa; palpitation; no albuminuria; cardiac murmur	12 months	Extreme anæmia; firm, yellow fat; ecchymosis of pia mater; obsolete tubercle of lungs; emphysema.
7	Idem (ibid.)	F., 40	Slight diarrhœa during lactation; excessive pallor; no albuminuria; dyspnœa; œdema	5 months	Excessive anæmia, with slight œdema; dilated stomach; slight ecchymosis.

TABLE B.—*One Hundred and Three selected Cases of Idiopathic Anæmia followed by Death and Autopsy, recorded since Addison's account of the Disease published in 1855.*

No.	Physician, with date of publication and reference.	Sex and age.	Antecedent conditions.	Symptoms.	Duration of disease.	Results of autopsy.
1	Wilks, 1857 ('Guy's Hosp. Rep.,' 3rd ser., vol. iii, p. 205)	F., 31	No hæmorrhage or diarrhœa. Menses regular. Had once been demented	Excessive pallor; cardiac murmur; vomiting and diarrhœa shortly before death; no leuchæmia	12 months	No wasting of body; characteristic fatty degeneration of heart; fatty liver and kidneys; ecchymoses [March, 1855].
2	Addison (ibid., p. 207)	M., 16	Occasional epistaxis. Diarrhœa lately	Extreme anæmia; improvement; relapse; no leuchæmia	12—15 months	Anæmia without wasting; fatty heart; liver and kidney less so; healed ulcers in colon and rectum.
3	Barlow (ibid., p. 208)	M., 52	Lupus in youth	Anæmia, dyspnoea, &c.; no emaciation	8 months	Extreme fatty degeneration of heart; fatty liver and kidneys; œdema.
4	Addison (ibid., p. 209)	F., 31	Recent parturition; previous epistaxis and diarrhœa	Cardiac murmur, and other signs of excessive anæmia	4 months	Extreme fatty degeneration of heart; fatty liver and kidneys; other organs healthy [May, 1853].
5	Wilks (ibid., p. 210)	F., 50	Lunatic for many years	Occasional vomiting; no hæmorrhage or diarrhœa; ingruvescent anæmia	6 months or less	Fatty degeneration of heart; fatty liver and kidneys; spleen, lymphatic glands, adrenals, semilunar ganglia, normal.
6	Idem (ibid., p. 211)	M., 30	Once hæmorrhage at stool	Dyspnoea, &c.; occasional pyrexia	4 months	Yellowish anæmia with œdema; heart, liver, and kidneys fatty; spleen 11 oz.
7	Lebert, 1858 (Wiener Med. Wochenschrift, No. 34, "Essentielle Anämie"), Zurich	M., 44	Frequent diarrhœa, &c.	Cardiac and venous murmurs; temporary improvement	6 months or more	Extreme anæmia; liver enlarged (? fatty); leucin and tyrosin in liver and other organs.
8	Bristowe, 1858 ('Path. Trans.,' ix, p. 432; 'Med. T. & Gaz.,' i, p. 267, "Fatal Anæmia")	M., 26	Occasional bilious attacks with vomiting, and once jaundice	Vertigo; vomiting; slight general jaundice; delirium	6 months	Marked anæmia with some œdema; Heart large; fatty degeneration of left ventricle; liver large, fatty; spleen large (16 oz.); no leuchæmia.

No.	Physician, with date of publication and reference.	Sex and age.	Antecedent conditions.	Symptoms.	Duration of disease.	Results of autopsy.
9	Leared, 1858 ('Path. Trans.,' ix, p. 438), "Death from Anæmia"	F., 11	No antecedent cause discoverable	Emaciation and intense anæmia; cardiac murmur; dyspnœa; vomiting; low temperature	7 months	Blood very pale and scanty; heart, fatty degeneration; spleen normal; no leuchæmia; red blood disks, very variable in size.
10	Wilks, 1859 ('Guy's Hosp. Rep.,' p. 108, "Simple or Idiopathic Anæmia")	F., 46	Occasional diarrhœa	Dyspnœa; œdema; cardiac murmur	6 months or more	Extreme anæmia with œdema; fatty degeneration of heart.
11	Cazenave, 1860 ('Journ. de Méd. de Bordeaux,' p. 53, quoted by Scheby Buch,) "Anémie Essentielle"	M., 21	Privation	Extreme anæmia; yellow tint without jaundice; œdema; weakness; palpitation; dyspnœa	?	Organs exsanguine; anasarca and serous effusion; no organic disease found.
12	Habershon, 1863 ('Lancet,' vol. i, pp. 518, 551, "Idiopathic Anæmia")	F., 40	Frequent vomiting; latterly almost daily	Excessive anæmia; no leuchæmia; great improvement; relapse of anæmia without vomiting	18 months or more	Extreme anæmia with great œdema; fatty degeneration of left ventricle.
13	Barelay and Dickinson, 1863 ('Path. Trans.,' vol. xiv, p. 141, "Fatal Anæmia. Fatty Heart")	M., 44	Privation; boils; weak intellect	Emaciation and excessive pallor; cardiac bruit	Above a year	General anæmia and œdema; heart pale, fatty degeneration; spleen small, 4 oz.; no leuchæmia; blood pale, scanty, fluid.
14	Wagner, 1864 ('Fett-metamorphose des Herzfleisches,' p. 158), Leipzig	F., 41	Half starved, strolling player; eczema	Ingravescent anæmia, with its effects; good appetite	?	No wasting; pallor and œdema; fatty heart and liver; pachymeningitis; œdema of lungs and pleurisy; small spleen; dark muscles.
15	? Troussseau, 1865 ('Clinique Méd.,'	F., 25	Suckling; starvation	Emaciation and pallor; pyrexia; no leuchæmia. Death from variola	3 months or more	General anæmia; heart small; liver enlarged and pale (? fatty); spleen large.

17	F., 25	After parturition; vomiting during pregnancy	Anasarca; cardiac murmur; pyrexia	6 weeks	General œdema and extreme anæmia; large fatty liver; heart soft; fatty kidneys.
17*	F., 24	Frequent parturition	Anæmia without wasting; diarrhoea; yellow colour; cardiac murmurs; dyspnoea.	3 months	Excessive anæmia; œdema of lungs large fatty liver; spleen normal (heart?)
18	F., 27	Occasional diarrhoea	Cardiac murmurs; temp. 100·4°; vomiting	About 12 months	No emaciation; extreme anæmia; ecchymoses of dura mater and of pleura; no leucæmia; heart and other organs normal.
19	F., 29	Pregnancy	Vomiting; œdema; cardiac murmur; delivery with little hæmorrhage; <i>transfusion</i> 150 cc.	2 months	General anæmia, with dropsy; heart "bright yellow colour;" liver also fatty; ecchymoses in stomach; spleen somewhat large.
20	F., 24	Pregnancy. Privation during war	Vomiting; delivery by version; <i>transfusion</i> 3 oz.	3 months	Anæmia; no wasting; "heart pale and spotted with yellow;" œdema; spleen enlarged.
21	F., 31	Healthy	Anæmia without wasting; œdema; cardiac and venous murmur; no retinal hæmorrhage; pyrexia; slight temporary paresis	7 weeks	No wasting; fatty degeneration of heart, liver, and kidneys; "spleen slightly enlarged."
22	F., 43	Healthy	Vomiting; anæmia; no pyrexia; chloasma; delirium	6 months	Well nourished; no leucæmia; blood stained yellow; adrenals small, but healthy.
23	M., 57	Icterus	Anæmia when the jaundice disappeared; vomiting; cardiac murmur; no pyrexia; no leucæmia	3 months	Ecchymosis of pericardium; fatty degeneration of heart; fatty liver; large spleen (marrow?).
24	F., 32	Puerperal flooding	Cardiac bruit; vomiting; slight pyrexia; <i>transfusion</i>	12 weeks	Anæmia; fatty degeneration of heart; capillary hæmorrhages in brain.

No.	Physician, with date of publication and reference.	Sex and age.	Antecedent conditions.	Symptoms.	Duration of disease.	Results of autopsy.
25	Pye-Smith, 1875 (Virchow's 'Archiv,' "Anæmia Idiopathica perniciosa")	M., 52	Healthy before. Rheumatism at 30	Slight diarrhoea; extreme anæmia; without wasting; heart normal; venous murmur; no leucæmia or pœciocytosis; temp. normal	6 months	Edema; heart fatty, valves normal; one or two infarcta in spleen; no other disease.
26	Idem (ibid.), lxx, p. 507, Guy's Hospital	M., 47	"Fever in youth" (?). Pallor and dyspnoea for several years; occasional diarrhoea	Ecchymosis on skin; edema; cardiac murmur; no leucæmia; no pyrexia; <i>phosphorus</i> ; occasional low temperature; traces of albumen in urine; coma vigil	In- definite	Edema and pleural effusion; fat; fatty degeneration of heart; fatty liver; numerous small calculi; post-mortem digestion of stomach.
27	Quinke, 1876 ('Volk- mann's Sammlung Kl. Vortr.,' No. 100, May, 1876, p. 810), Bern	F., 34	Ill fed. Diarrhoea	Cardiac murmur; edema; diarrhoea; slight occasional albuminuria; retinal hæmorrhage; no leucæmia; no pyrexia; <i>arterial transfusion</i> (100 cc. human blood)	3 months	Anæmia and edema; heart?, aorta small; infarcta in kidney and spleen; diphtheritic colitis.
28	Idem (ibid., p. 812), "Perniciöse Anämie"	F., 11	Doubtful	The preceding year admitted with great anæmia and retinal hæmorrhage; went out well; relapse; retinal hæmorrhage; diarrhoea; pyrexia; pœciocytosis	2 years or more	Anæmia and dropsy; ecchymoses of pericardium.
29	Idem (ibid., p. 814)	M., 45	Healthy	Anæmia; edema; epistaxis; "slight icterus;" cardiac murmur; retinal hæmorrhage; slight pyrexia; pœciocytosis	4 months	Anæmia without wasting; fatty degeneration of left ventricle.
30	Idem (ibid.)	F., 34	"	Anæmia and wasting; ecchymosis on skin and in retina; cardiac murmur; vomiting; slight fever; epistaxis; pœciocytosis; <i>transfusion</i> (250 cc. human blood)	Several months	Anæmia; fatty degeneration of heart; slightly swollen spleen; ecchymoses of pia, pericardium, stomach, bladder, and pancreas.
31	Lebert, 1876	F.,	No cause ascertained	Anæmia and edema; cardiac and	3 months	Fatty degeneration of heart; organs

33	M., 48	Scheby-Buch, 1876 (‘Deutsches Arch. f. Kl. Med.,’ xvii, 467)	Large chronic ulcers of legs	Anæmia; no cardiac murmur; re- tinal hæmorrhage; no leucæmia	A year or more	Excessive anæmia; œdema of menin- ges; heart small, pale, and flac- cid; spleen moderately enlarged.
34	F., 60	Idem (ibid.), “Essen- tielle Anämie”	Healthy	Anæmia without wasting; no mur- mur; no leucæmia	18 mos. or more	Yellow fat; retinal hæmorrhage; spleen swollen; marrow normal.
35	M., 24	Kussmaul and Maier, 1877, Strassburg (quoted by Lépine)	”	Pallor and weakness; epistaxis; py- rexia	7 weeks	Well nourished; œdema; fatty heart?; and liver and kidneys; ecchymoses of stomach and muscles.
36	F., 34	Lépine, 1876 (‘Revue mensuelle de Méd. et de Chir.,’ p. 129)	Pregnancy	Pallor; œdema; diarrhœa; cardiac murmur; no leucæmia	8 months	Wasting; fatty degeneration of heart; œdema; marrow “pale and somewhat gelatinous.”
37	F., 28	Ferrand, 1876 (ibid., p. 730)	Diarrhœa; parturi- tion	Intense anæmia; pyrexia; cardiac murmur; <i>transfusion in extremis</i>	6—8 months	Anæmia and œdema; heart pale; liver fatty; spleen 360 grammes; marrow normal.
38	M., 21	Habershon, 1876 (‘Med. Times & Gaz.,’ March, 1876, p. 249)	Occasional epistaxis	Pallor; occasional vomiting; arterial and venous murmurs; no pyrexia; <i>transfusion</i> 6 oz., human blood	2 months	Anæmia and œdema; fatty degenera- tion of heart; ecchymoses of peri- cardium.
39	M., 53	Moxon (‘Lancet,’ 1877, vol. i, p. 642)	Similar symptoms for 2 months, ten years ago	Ingravescent anæmia; cardiac mur- mur; pyrexia; pœciocytosis; no retinal hæmorrhage; <i>phosphorus</i>	5 months	Anæmia and œdema; organs nor- mal, except small old abscess in one lung; no wasting.
40	M., 51	Stricker, 1877 (‘Perni- ciöse Anämie,’ ‘Charité Annalen,’ ii, p. 289. Quoted by Eichhorst)	Single ulcer of leg	Pallor without wasting; ecchymoses of skin; spongy gums; cardiac and venous murmur; retinal hæ- morrhage	6 weeks	Well nourished; general anæmia; heart not fatty, dilated; ecchymosis in pia and cerebellum and in colon.
41	M., 36	Rosenstein, 1877 (‘Perniciöse Anämie,’ ‘Berliner klin. Wochen- schr.,’ No. 9). Leyden	Enterica 6 months ago	Diarrhœa and œdema; cardiac mur- mur; blood normal; pyrexia; re- tinal hæmorrhage	6 weeks	Heart not fatty; granular degenera- tion; liver and spleen enlarged; marrow normal.

No.	Physician, with date of publication and reference.	Sex and age.	Antecedent conditions.	Symptoms.	Duration of disease.	Results of autopsy.
42	Risdon Bennett, 1877 ('Med. Times and Gaz.,' i, p. 471)	M., 42	Healthy before	Ingravescent anæmia; cardiac murmur; no pyrexia	5 months	Abundant fat; pleural effusion (without pleurisy) and general œdema; fatty degeneration of left ventricle.
43	B. Bramwell, 1877 ('Edin. Med. Journ.,' xxiii, p. 408)	M., 34	Never robust; no cause of illness	Retinal hæmorrhage; pœcilocytosis; vomiting and diarrhœa; irregular pyrexia; final pneumonia	2 years	Fatty heart, liver, and kidneys; thyroid and spleen large; hepatised lungs.
44	Idem (ibid.), "Idiopathic or Pernicious Anæmia"	M., 28	Had yellow fever	Retinal hæmorrhages; diarrhœa; no fever	7—8 months	Fatty heart, liver, and kidneys; slight intestinal ulceration
45	Purser, 1877 ('Dublin J. of Med. Sci.,' Nov., 1877, p. 405)	F., 60	Good health until a "strain"	Pallor without wasting or œdema; no pyrexia; vomiting; cardiac and venous murmurs; pains; pœcilocytosis and microcytosis; petechiæ	no 2 months	Bright yellow fat; no dropsy; fatty degeneration of heart; retinal hæmorrhage.
46	Burger, 1876 ('Berl. kl. Wochensch.,' Aug., 33 and 34), quoted by Lépine	F., 25	Never strong; recent abortion	Pallor; œdema; no leucæmia; urine normal; retinal hæmorrhage; diarrhœa before death	Several months	Well nourished; pachymeningitis hæmorrhagica; œdema and ascites; fatty degeneration of heart and liver; also of gastric and intestinal tubules.
47	Quinke, 1877 ('Deutsches Archiv,' xx, p. 1—31)	F., 47	Poor living	Ingravescent anæmia; blood? <i>transfusion</i> (into artery)	12 mos. or more	Wasting; hæmorrhagic pachymeningitis; œdema and serous effusion in chest; heart fatty; spleen 4½ oz.; scar in apex of one lung.
48	Biermer, 1877, Zürich (H. Müller, 'Die progr. Pern. Anämie,' p. 19)	M., 29	Healthy before	Rapidly ingravescent anæmia; cardiac murmur; swollen spleen; pyrexia; vomiting; lethargy	7 weeks	Anæmia; œdema; slight splenic enlargement. [1866.]
49	Idem (ibid., p. 23)	F., 52	Diarrhœa, &c., for five years	Cardiac and venous murmurs; blood normal; pyrexia; retinal hæmorrhage	6 months	Anæmia and general dropsy; fatty degeneration of heart. [1867.]
50	Idem (ibid., p. 24)	F., 40	Well till last pregnancy, which ended in abortion	Cardiac and arterial murmurs; dyspnoea; œdema; pyrexia	—	Anæmia and dropsy; fatty heart and liver. [1868.]

53	Idem (ibid., p. 35)	F., 35	Healthy	Cardiac murmur; pyrexia; retinal hæmorrhage; exophthalmos	11 mos.	Dropsy; fatty heart; small thyroid; ecchymoses of pia, pleura, stomach, and other organs.
54	Idem (ibid., p. 31)	F., 35	No cause obvious	Cardiac murmur; pyrexia; diarrhoea	2 months	General dropsy; fatty degeneration of heart.
55	Idem (ibid., p. 59)	F., 24	Chlorosis; epistaxis	Pyrexia; ecchymoses; cardiac murmur	6 mos. ?	General dropsy; heart pale; no fatty degeneration.
56	Idem (ibid., p. 68)	F., 18	Chlorosis	Cardiac murmurs (systolic and diastolic); pyrexia; retinal hæmorrhage	?	Anæmia and cedema; fatty degeneration of heart; ecchymoses
57	Idem (ibid., p. 64)	M., 46	Healthy	Cardiac bruit; pyrexia; diarrhoea; final pneumonia	2 months	Dropsy; fatty degeneration of heart; recent hepatisation of one lung.
58	Idem (ibid., p. 65)	F., 30	Since parturition	Cardiac bruit; ecchymoses; vomiting; final erysipelas of face	?	Dropsy; hæmorrhage of dura mater.
59	Idem (ibid., p. 65)	M., 55	Healthy	Edema; dyspnoea; cardiac bruit	4 mos. ?	Edema; fatty heart.
60	Idem (ibid., p. 77)	F., 29	Flooding; diarrhoea	Cardiac bruit; vomiting and diarrhoea	10 mos.	No cedema; wasting; fatty degeneration of heart; ecchymoses of pia.
61	Idem (ibid., p. 81)	F., 42	Since parturition	Cardiac and arterial murmurs; pyrexia; retinal hæmorrhage	10 mos.	Fatty heart; ecchymoses of dura; peri- and endocardium, and pleura; capillary hæmorrhage in cerebrum and bulb; enlarged spleen.
62	Idem (ibid., p. 88)	F., 32	Very healthy	Cardiac and arterial murmurs; pyrexia; retinal hæmorrhage; cedema; diarrhoea	4 months	Fatty heart; ecchymoses in brain, dura, and retina; cedema of lungs.
63	Idem (ibid., p. 89)	F., 23	Flooding 10 days after parturition; weak before	Cardiac murmur; pyrexia; vomiting; cedema; retinal hæmorrhage	4 months	Slight general dropsy; fatty heart; fatty liver and kidneys; ecchymoses in brain and slightly in liver; 90 small gallstones; no jaundice.
64	Idem (ibid., p. 110)	F., 35	Similar illness after parturition 4 years before; this began during pregnancy	Wasting; cardiac murmur; pyrexia; retinal hæmorrhage	3 months	Fatty heart; ecchymoses of dura, pia, and cerebellum; also of pericardium; recent pneumonia.
65	Idem (ibid., p. 114)	F., 31	Perfectly healthy	Diarrhoea; general cedema; arterial murmurs; no pyrexia; retinal hæmorrhage; ecchymoses	5 months	No fatty degeneration of heart; general dropsy; ecchymoses in brain and cord.

No.	Physician, with date of publication and reference.	Sex and age.	Antecedent conditions.	Symptoms.	Duration of disease.	Results of autopsy.
66	Idem (<i>ibid.</i> , p. 122)	F., 32	Puerperal flooding	Cardiac murmur; œdema; pyrexia; retinal hæmorrhage	6 weeks	General dropsy; fatty degeneration of heart; ecchymoses of dura, brain, and pericardium; cystitis.
67	Idem (<i>ibid.</i> , p. 142)	F., 26	Stomatitis and diarrhoea	Cardiac murmur; œdema; retinal hæmorrhage; attacks of temporary paralysis	3 years	Fatty heart; œdema; capillary hæmorrhage in corona radiata and in cerebellum.
68	Idem (<i>ibid.</i> , p. 154)	F., 39	Not ascertained	Cardiac murmur; dropsy; retinal hæmorrhage; no leucæmia; pyrexia	?	Fatty heart; œdema; ecchymosis of pia and brain.
69	Huguenin, Zürich (Müller, <i>loc. cit.</i> , p. 236) Cf. Table C., No. 8	F., 35	Earlier chlorosis; same symptoms a year before; recovery	Anæmia without wasting; cardiac bruit; pyrexia; no retinal hæmorrhage; improvement and relapse	10 mos.	Fatty heart, liver, and kidneys; fatty degeneration of diaphragm; capillary hæmorrhage of brain; dropsy.
70	Idem (<i>ibid.</i> , p. 241)	F., 35	Excellent health until puerperal hæmorrhage	Diarrhoea and vomiting; no murmurs; œdema; retinal hæmorrhage	6 months	Dropsy; wasting; no fatty degeneration of heart; ecchymoses of brain and stomach.
71	Idem (<i>ibid.</i> , p. 242)	F., 36	Excellent health	Diarrhoea; œdema; cardiac murmur; no pyrexia	9 months	Dropsy; wasting; fatty degeneration of heart.
72	Idem (<i>ibid.</i> , p. 243)	F., 42	Good health	Dropsy; cardiac murmur; retinal hæmorrhage; diarrhoea; pyrexia	7 months	Dropsy; fatty degeneration of heart.
73	Idem (<i>ibid.</i> , p. 243)	F., 39	Good health; ill-fed	Hæmatemesis; ingravescant anæmia; cardiac murmur; pyrexia; retinal hæmorrhage; poecilocytosis; no leucæmia; urea diminished	18 mos.	Ecchymoses of brain, pia, and stomach; fatty degeneration and some dilatation of heart.
74	Idem (<i>ibid.</i> , p. 245)	F., 8	Good health; well-fed	Vomiting; diarrhoea; increasing anæmia; œdema; cardiac murmur; retinal hæmorrhage; slight pyrexia; no leucæmia; no albuminuria	7 months	Excessive anæmia; fatty degeneration of heart; fatty liver; ecchymoses of pericardium only; small spleen; lungs normal, except dark spots of old minute apoplexies.
75	Idem (<i>ibid.</i> , p. 246)	M., 54	Healthy	Vomiting and diarrhoea; increasing weakness and anæmia; general dropsy; cardiac murmur; normal blood and urine; no retinal hæmorrhage	12 mos.	Dropsy; slight fatty degeneration of heart; no hæmorrhage but in retina; spleen somewhat enlarged.

77	Idem (ibid., p. 247)	M., 66	Healthy	blood and urine; retinal hæmorrhage; pyrexia	6 months	Anæmia and œdema; fatty degeneration of heart; no hæmorrhage.
78	Barlow (Taylor, 'Guy's Hosp. Rep.,' 1878, 3rd ser., vol. xxiii, p. 190), "Idiopathic Anæmia,"	M., 22	Good health; temperature	Anæmia; weakness; dyspnoea; cardiac bruit; epistaxis; albuminuria	5 weeks	Lungs and other organs healthy; blood scanty, very little clot; liver and kidneys pale but healthy; spleen small. [1864.]
79	Wilks (ibid., p. 190)	F., 52	Domestic troubles	Pallor; prostration; no albuminuria; fœtid odour	3 months	General œdema; watery blood; brain, lungs, liver, kidneys, adrenals very anæmic; spleen 8 oz.; post-mortem digestion of stomach; heart pale, fatty degeneration. [1867.]
80	Idem (ibid., p. 191) "Idiopathic Anæmia"	M., 51	Good health	Pallor and œdema; no cardiac murmur; no albuminuria	10 mos.	Fat; large ecchymoses of pia; old pleuritic adhesions; obsolete phthisis of one lung; fatty degeneration of heart; blood pale, non-coagulating. [1868.]
81	Idem (ibid., p. 194) "Idiopathic Anæmia"	M., 68	"	Diarrhœa; vomiting; pallor; no pyrexia; no bruit; delirium before death; no leuchæmia	18 mos.	Organs anæmic; fatty degeneration of heart, and recent pneumonia of base of one lung. [1870.]
82	Moxon (ibid., p. 199) "Idiopathic Anæmia"	F., 32	Ulcer of leg, but good colour and flesh	Lemon-yellow colour; wasting; œdema; ecchymoses of skin; pyrexia; vomiting; bleeding gums	7 months	Edema; ecchymoses; fatty degeneration of heart. [1874.]
83	Wilks (ibid., p. 201)	M., 47	Never strong	Pallor; wasting; no œdema; improvement. <i>Iron</i> ; relapse	18 mos.	Anæmia and œdema; no fatty degeneration of cardiac muscle; spleen, kidneys, and adrenals normal; recent pleurisy.
84	Habershon (ibid., p. 206)	F., 15	Never strong; eczema shortly before anæmic symptoms	Anæmia; cardiac and venous murmurs; epistaxis; vomiting; pyrexia; skin darkened in places	2 months or more	General anæmia; wasting œdema; fatty degeneration of heart; other organs normal, except one obsolete calcareous bronchial gland.
85	Gull & Dickinson, 1878 ('Path. Trans.,' xxix, p. 379), "Idiopathic Anæmia"	M., 68	Healthy and strong	Gradual anæmia, rapidly increasing, without pyrexia, vomiting, or diarrhœa; no leuchæmia; red disks, partly small and irregular	6 months	Well nourished; excessive anæmia; little œdema; fatty degeneration of heart; single serous cyst in liver; four gall-stones (no jaundice); spleen soft; adrenals normal.

No.	Physician, with date of publication and reference.	Sex and age.	Antecedent conditions.	Symptoms.	Duration of disease.	Results of autopsy
86	S. Mackenzie, 1878 ('Lancet,' ij, p. 13)	M., 10	Healthy before	Pallor; cardiac murmur; retinal hæmorrhage; optic neuritis; epistaxis; vomiting; pœcilocytosis; pyrexia	5 months	Yellowsubcutaneous fat; fatty degeneration of heart; spleen, lympharia, adrenals, and marrow normal.
87	Eichhorst, 1878 ('Die pr. pern. Anämie,' p. 121), Berlin	M., 24	Healthy	Excessive pallor, without wasting; venous murmur; pœcilocytosis; systolic cardiac murmur; delirium	6 weeks	Well nourished; anæmia and œdema; fatty heart; marrow normal.
88	Idem (ibid., p. 128)	M., 42	Fever in youth; served as a soldier	Weakness and pallor without wasting; retina normal; enlarged spleen; pœcilocytosis; death by coma; loss of hair and cadaveric odour	7 months	Edema; abundant yellow fat; fatty degeneration of heart; ecchymoses in pericardium, meninges, and retina; also in pelvis renis; spleen enlarged; marrow normal.
89	Idem (ibid., p. 144)	F., 44	No cause ascertained	Wasting and pallor; œdema; no retinal hæmorrhage; cardiac, arterial, and venous murmurs; pœcilocytosis; loss of hair and same odour before death	8 months	Edema; fatty and dilated heart; hæmorrhage in pia, in duodenum, and in pelvis renis; yellow softening of brain; marrow normal.
90	Idem (ibid., p. 156)	F., 37	Good health	Pallor and œdema; no bruit; retina normal; temperature low; pœcilocytosis	5 months	Dropsy; fatty heart; hæmorrhage of stomach, pleura, &c.; spleen and mesenteric glands enlarged; marrow normal.
91	Idem (ibid., p. 165)	F., 29	Rheumatism several years before	Pallor and œdema; arterial and venous murmurs; retinal hæmorrhage; microcytosis; no leucæmia	4 months	Abundant sulphur-yellow fat; fatty degeneration of heart; spleen enlarged and mesenteric glands caseous.
92	Kahler, 1880 ('Jahresbericht,' ii, p. 249), Bohemia	M., 12	No cause	Ingravescent anæmia; dyspnoea and other sequelæ; blood corpuscles large	?	Fatty degeneration of heart; other organs exsanguine.
93	Mitchinson, 1881	F.,	Good health	Pallor; œdema; diarrhœa and vomit-	12	Yellow tint; slightly wasted; no or-

95	Allbutt, 1881 (reported by Barrs, 'Brit. Med. Journ.,' p. 627)	M., 43	Healthy	palliation; cardiac and venous murmurs; retinal hæmorrhage, with optic neuritis; slight pyrexia	Nearly 2 years	Fairly nourished; earthy tint; oedema of lungs; obsolete apex scar; heart 13 oz., not fatty; spleen 8 oz.; gastritis.	large (16 oz.), early fatty degeneration; aorta small; spleen small.
96	Greenhow (reported by Coupland, 'Brit. Med. Journ.,' 1881 i, p. 550)	F., 29	Fairly well	Pallor; dyspnoea; vomiting; diarrhoea; cardiac and venous murmurs; pyrexia; slight pigmentation	7 weeks	Not emaciated; oedema; heart pale and flabby.	
97	Cayley (ibid., p. 550)	M., 43	"	Pallor and oedema; pyrexia; transient aphasia and paresis	8 weeks	Dropsy; ecchymoses in retina, none found in brain; fatty degeneration of heart; old pericardial adhesion.	
98	Idem (ibid., p. 551)	M., 7	Never strong	Pallor; no wasting; oedema; cardiac murmur; retinal hæmorrhage; pyrexia	5 weeks	Ecchymoses; fatty degeneration of heart; fatty liver, kidneys, &c., normal.	
99	Pye-Smith [1871], (unpublished)	M., 24	Fairly well	Pallor; cough; dyspnoea; pyrexia; epistaxis; bed sore, which healed	4 months	Oedema; fatty degeneration of musculi papillares; ecchymoses; sanguineous effusion in pleura; oedema pulmonum; spleen not enlarged; slight enlargement of lymphatic glands.	
100	Idem (present case), <i>vide</i> supra, p. 219	M., 46	Dyspeptic from boyhood, with occasional diarrhoea	Pallor without wasting; pyrexia; no retinal hæmorrhage. <i>Arsenic</i> ; improvement; relapse; <i>transfusion</i>	2 years	Excessive anæmia, with good amount of fat; oedema; ecchymoses; fatty heart; spleen soft; marrow normal.	
101	F. Taylor [1878], (unpublished)	M., 38	Dysentery in China 15 years ago	Anæmia; icterus; cardiac murmur; delirium; improvement; relapse; of severe pœciocytosis	6 months	Extreme anæmia; fairly nourished; obsolete scar at one apex; heart 15 oz; slight fatty degeneration; colon normal; liver 62 oz.; spleen 16 oz., soft.	
102	Carrington, 1882 ('Lancet,' Feb. 3)	M., 21	No history; Norwegian sailor	Extreme pallor and dyspnoea; syncope; oedema; no bruit; retinal hæmorrhage; pœciocytosis; slight pyrexia; vomiting	Probably short	Bloodless, cedematous organs; abundant fat; ecchymoses of pericardium; spleen not swollen; marrow normal.	

TABLE C.—Twenty cases of Recovery from Idiopathic and profound Anæmia.

No.	Author.	Sex and age.	Antecedents.	Symptoms.	Duration, treatment, and event.
1	Quinke (‘Volkmann’s Vortr.’ 1876)	M., 35	Good health; said to have been a miser, and lived poorly	Pallor and œdema; cardiac murmur; micro- cytosis; retinal hæmorrhage; tempera- ture?	Ten months ill; six weeks’ treatment, soda and gentian, then iron; incomplete re- covery; well by last account.
2	Idem, 1877 (‘Deut. Archiv,’ vol. xx, p. 1)	F., 33	Poverty and over- lactation	Palpitation; dyspnoea; waxy aspect; cardiac murmur; pœciocytosis; retinal hæmor- rhage; blood corps. 143,000 per cubic mm., raised on recovery to 1,234,000	Eight months ill; transfusion, 185 c.c. de- fibrinated human blood; perfect recovery.
3	Idem (ibid., p. 3)	M., 43	Twice ill in the same way before (?); no cause obvious	Pallor; dyspnoea; palpitation; cardiac mur- mur; spleen normal; retinal hæmorrhage; petechiæ of skin; pœciocytosis; blood corps. 539,000 per cubic mm.	Ill two months; transfusion, 50 c.c. human blood into radial artery; recovery after four months’ treatment; blood corps. 2,998,800.
4	Idem (ibid., p. 11)	M., 42	Fair health until lately; similar at- tacks Sept., Oct., ’73, June, Sept., ’76	In 1877, pallor with pœciocytosis and re- tinal hæmorrhage; pyrexia	Recovery in two months without steel, ar- senic, or transfusion. A chronic and not very severe case.
5	Idem (ibid., p. 14)	F., 40	Good health	Moderate anæmia; slight cardiac murmur; doubtful pœciocytosis; 1,689,000 in cubic mm.; retinal hæmorrhage; temperature?	Nine months ill; rapid improvement and recovery in six weeks under food and iron. The slightest case of anæmia with retinal hæmorrhage.
6	Bierner (Müller, p. 85)	F., 19	Chlorosis; epistaxis	Excessive pallor and œdema; cardiac mur- mur; retinal hæmorrhage; pyrexia	Jan. 1870 to Oct. 1873; iron; well in 1874.
7	Idem (ibid., p. 95)	F., 36	Chlorosis	Ingravescent anæmia; venous and cardiac murmurs; retinal hæmorrhage; pyrexia	Eighteen months ill; stimulants; well a year after discharge.
8	Idem (ibid., p. 101)	F., 34	”	Anæmia and œdema; cardiac murmur; re- tinal hæmorrhage and petechiæ on skin; large spleen; occasional fever; slight hemiplegia	Two years ill; a year later readmitted with the same symptoms, and died. See No. 69 in Table of Fatal Cases.

11	p. 237) Bramwell ('Edin. Med. Journ., Nov., 1877)	M., 20	vomiting and pallor for several months Yellow fever shortly before	vascular murmurs; epigastric tumour, supposed to be <i>carc. pylori</i> ; this gradually disappeared; pyrexia Pallor; yellow tint; œdema; no wasting; retinal hæmorrhage; poecilocytosis; pyrexia; vomiting; diarrhœa	Ill four months and a half; lime-juice and arsenic; convalescent in three months.
12	Idem (ibid.)	M., 43	Good health	Excessive anæmia; yellow tint; wasted; poecilocytosis; paresis; numbness, &c.	Ill two months and a half; same treatment as last case; well after four months.
13	Idem (ibid.)	M., 38	"	Pallor; yellow tint; no wasting; retinal hæmorrhage; poecilocytosis; pyrexia; vomiting and diarrhœa	Ill seven months; no improvement under iron and cod-liver oil; arsenic; perfectly well in two months.
14	Idem (ibid.)	F., 31	Pregnancy	The same yellowish colour; anæmia and œdema; no wasting; retinal hæmorrhage; poecilocytosis; vomiting and diarrhœa; pneumonia	Ill four months; recovery under steel medicine.
15	Eichhorst (loc. cit., p. 180)	M., 37 or 57?	Enterica (?) ten years ago; good health	Pallor; œdema; dyspnoea; vomiting; defæcivum capillorum; venous murmur; blood pale; disks large (8-9 μ) and pale; also some small, round and dark (3 μ)	Nine months ill; benefit from country air and good living after leaving the hospital in Berlin.
16	Finny ('Brit. Med. Journ., Jan. 3, 1880)	M., 27	Good health	Pallor; weakness; diarrhœa; sallow, waxy hue; œdema; venous murmur; no emaciation; pyrexia (104.5); poecilo- and microcytosis; hair thin	Ill four years; iron found useless; arsenic; steady improvement; complete recovery; blood normal; hair thick and dark.
17	Idem (ibid.)	M., 48	—	Extreme pallor; cardiac and venous murmurs; no pyrexia, poecilocytosis; vomiting	Ill over a year; arsenic; slow improvement; well a year afterwards.
18	Broadbent (Brit. Med. J., Sept. 25, 1880)	F., 42	Good health	Pallor; yellow tint; venous murmur; pyrexia; poecilocytosis; 500,000 red corpuscles per cubic mm.	Five months ill; three months treatment with arsenic.
19	Mitchinson ('Lancet,' Jan. 1, 1881)	F., 24	—	Pallor; œdema; diarrhœa; vomiting; spleen, &c., normal; poecilocytosis; hæmoglobin one-tenth of normal; pyrexia	Steel and arsenic; hæmoglobin increased to 40 and then to 75 p.c. of normal; went out nearly well.
20	Pye-Smith (supra, p. 277)	M., 53	Good health	Excessive anæmia; no wasting; retinal hæmorrhage, &c.	Six months ill; improvement; relapse; arsenic; complete recovery after about eighteen months.

POISONING BY ACONITINE.

(CASE OF REG. *v.* LAMSON.)

By THOMAS STEVENSON, M.D.

THE great interest shown by the public and by the profession in the trial of George Henry Lamson, a medical practitioner, for the murder by poison of his brother-in-law, Percy Malcolm John, in 1881—the rareness of poisoning by the alkaloids of monkshood—the novelty of some of the points raised—and, I may add, the moral indignation exhibited towards myself by a number of well-meaning enthusiasts, as displayed in their letters addressed to me, in consequence of my so-called vivisection experiments with reference to the above case,—are considerations which have induced me to place before the readers of these Reports as concise an account as possible of the above case. It is one which will doubtless hereafter be quoted as a leading case in forensic medicine.

Percy Malcolm John was the youngest of a family of five children, one of whom died in 1873, and another in 1879. One of the two surviving daughters married George Henry Lamson, a medical man, of American (U.S.) nationality; and the other a Mr. Chapman. Percy at the time of his death, which occurred on December 3rd, 1881, was very nearly nineteen years of age, and had for the three years preceding been an inmate of Mr. Bedbrook's Boarding School at Wimbledon. He suffered from old spinal curvature and paralysis of the lower limbs, so that although above the pelvis he had the development of a

powerfully built man, the lower portion of the body and the lower limbs were not more developed than those of a child. He was unable to walk, but was able to move himself about in a wheel-chair, and to get up and downstairs by the aid of his arms with a sidelong movement. Usually, however, he was carried up and downstairs on the back of a school-fellow. His general health was good; and although in the latter part of 1881 it was thought that the spinal curvature was increasing, no effects of this upon his general health were apparent. He required no medical attendance, or only of the most trivial description, and he was of a cheerful disposition. Had he attained his majority he would have been entitled to a sum of about £3000, of which, by his death, one half passed into the hands of his sister, Mrs. Lamson. As there was no regular settlement of her property, made at her marriage, Mrs. Lamson's share would at Percy's death pass into her husband's hands.

In the early part of the year 1881, Lamson was in pecuniary difficulties, which necessitated his leaving Bournemouth, where he was heavily in debt to several persons; and he sailed from Liverpool to New York on April 7th, returning by the same vessel on July 2nd, 1882. Whilst in the United States he appears to have made a first attempt to poison his brother-in-law. He sent over from New York for the use of the latter a box containing eleven or twelve pills, with a letter stating that this form of pill had been found serviceable in cases of spinal disease. Percy John took one of these pills, which were stated to contain quinine, one night in the presence of Mr Bedbrook. He was so unwell afterwards that he refused to take another pill, and it was supposed that the remaining ten or eleven pills had been destroyed; but after John's death a box of pills similar to that sent over was found in the deceased's school box. In it were two pills carefully wrapped up separately in tin-foil, as if to preserve them from damp during a sea-voyage. These pills contained quinine and aconitine, of which latter alkaloid one contained nearly half a grain, and the other a smaller quantity. The alkaloid extracted from these pills was as potent as English aconitine.

On August 27th, 1881, Percy went with Mr. and Mrs. Chapman to stay at Clarence Villa, Shanklin. G. H. Lamson, who was staying in the island with his father at Ventnor, met them

on their arrival at the station, and accompanied them to the rooms he had secured for them. He stated that on the 29th (Monday) he was leaving for America, and would call on them on that day.

Mr. and Mrs. Chapman were out when he called. He walked through the open French window from the lawn into the room where Percy was sitting. It appears that he gave Percy what purported to be either a quinine powder or a quinine pill. It was taken by the youth, but at what time of the day is unknown. After he had taken it he was seized with illness. He retired at 9.30 p.m., earlier than usual, and when he was in bed he complained of feeling paralysed all over, and appeared to be very unwell. In the night, or towards morning, his bowels were greatly relaxed, and again at 6 a.m. He got better, however, after breakfast.

It may be mentioned in connection with this incident, which was doubtless a second attempt on the life of Percy John, that on Sunday, August 28th, 1881, Lamson went to the shop of Mr. Smith, a chemist and druggist in Ventnor, and purchased a grain of aconitine which he took away with him.

On October 13th, 1880, nearly fourteen months before Percy John's death, and about ten months before his illness at Shanklin, Lamson had purchased of a chemist and druggist at Ventnor twelve quinine powders, of a grain and a half each. Six of these powders were found in John's box after his death, with fourteen other quinine powders, three of the latter being mixed with aconitine. These fourteen powders were numbered in series 7 to 20, both numbers inclusive, and were of variable weights, ranging from 0.62 to 1.26 grains; Nos. 16, 17, and 19, alone contained aconitine, identical with Morson's English aconitine. No. 16 contained the largest quantity of this alkaloid, viz. 0.83 grain. How, when, or where these powders came into Percy John's possession is not known; but it was thought that they might have been given to him by Lamson at Shanklin on the occasion above mentioned.

On November 11th, 1881, Lamson went to the shop of Messrs. Bell and Co., Oxford Street, and had a prescription made up of morphia and atropine, for hypodermic injection. On the 16th he called again, and asked for five grains of digitalin, saying that he wanted it for external use. The preparation in

stock was found to be discoloured, and the assistant thinking it might not be pure declined to supply it. Soon after November 20th, Lamson again called, and asked for a grain of aconitine for external use. This the assistant declined to supply, and advised him to go somewhere where he was better known. On the 24th he went to Messrs. Allen and Hanbury's, Lombard Street, and asked for two grains of aconitine, and being asked his name he wrote an order for the alkaloid and signed it as G. H. Lamson, M.D., Bournemouth, Hants. That being a name and address in the Medical Directory the aconitine was supplied, the wholesale price, fifteenpence per grain, being charged.

On December 2nd, 1881, Lamson packed up his luggage at his hotel in London, went with a friend named Tulloch to Waterloo Railway Station, left his luggage there, and went with Tulloch to Wimbledon, taking a hand-bag with him. Leaving Tulloch at a public house, he professed to have visited his brother-in-law and to have had a conversation with Mr. Bedbrook, the master; and he told Tulloch that Percy was growing worse, his spinal curvature increasing, and that he would not live long. The visit and conversation were fictitious; and it may be surmised from subsequent events, that Lamson had gone to Wimbledon with the intention of poisoning his brother-in-law, but that on this occasion his courage failed him at the last moment.

On Saturday, December 3rd—a half holiday—Percy dined at about 1 p.m.; boiled rabbit and onion sauce forming part of his dinner. This was followed by tea and bread and butter at 5.30 p.m. At 6.55 p.m. his brother-in-law Lamson unexpectedly arrived to pay, as he said, a farewell visit before taking his departure for Paris and Florence. Lamson was shown into the dining-room, an apartment about 16 feet square, with a lighted gas chandelier over the table. The prisoner seated himself, and next to him—not a yard distant—was seated Percy, who was carried in to see his relative. Five or six feet from them Mr. Bedbrook remained standing. A decanter of sherry wine was placed before Lamson, and he was assisted to a large glassful. The remark was made by Lamson: "Well, Percy, old boy, how fat you are looking." Lamson produced from a bag a Dundee cake and some sweetmeats; and the cake was cut by Lamson with his penknife and distributed to the party. He also asked for sugar to put into the sherry, and powdered white sugar was

brought by the matron. At 7.15 p.m. Lamson remarked, "Oh, by the way, Mr. Bedbrook, when I was in America I thought of you and your boys, and what excellent things these capsules would be for your boys to take nauseous medicines in." He then produced from his bag two boxes of gelatine capsules, such as are much used in the United States for the administration of medicaments by the mouth and rectum. They were composed of translucent gelatine, and consisted of two small, thimble-shaped parts, one a little larger than the other, so that when one part was slid over the other, a small box not unlike a rifle bullet in shape was formed, in which any medicine might be enclosed. Mr. Bedbrook examined them, and swallowed one at Lamson's request, in order to see how easily they could be taken. The boxes containing the capsules were half empty; one had been pushed to Mr. Bedbrook, the other remained immediately in front of Lamson, who took the lids off both boxes. Whilst the master was examining a capsule, he noticed Lamson filling another, which he held between his fingers, with sifted sugar from the basin, using for this purpose a spade sugar spoon. Lamson gave the capsule a shake forward, as he said, "to bring the sugar down to one end," and he then handed it to his brother-in-law, saying, "Here, Percy, you are a swell pill-taker; take this and show Mr. Bedbrook how easily this may be swallowed." The youth put the capsule into his mouth, and swallowed it. Lamson then said, "I must be going now." It was then 7.20 p.m. He left to catch the 7.21 p.m. train, the house being only a minute's walk from the station, in order to catch the 8 p.m. continental train from London Bridge Station. The administration of poison was thus ascertained to have taken place between 7.15 and 7.20 p.m. Not long after, at 7.45 or 7.50, Percy complained of heart-burn; and again, about five minutes later, added that he felt as he had felt in Shanklin on August 29th, 1881, after Lamson had given him a quinine pill or powder. He said he should like to go to bed. Shortly after this, about 8 p.m., he was carried upstairs into the bath-room where he vomited; and about a quarter of an hour later he was taken to his bed. In the bath-room he said that his skin felt all drawn up, and also that his mouth was very painful. At 8.30 he was vomiting violently, and was seemingly in great pain. He threw himself about most violently, and

complained that his skin felt drawn and his throat closing up. At 8.55 p.m. he was seen by Dr. O. W. Berry. He was then lying on his bed partially undressed. He complained of great pain in the region of the stomach, that the skin of his face felt drawn, of a sense of constriction in the throat, and inability to swallow. He was retching and vomiting a small quantity of dark-coloured fluid. White of egg beaten up with water was given, and of this he was able to swallow a little; and hot linseed poultices were applied to the region of the stomach. He was so restless and violent, throwing himself about, that it required more than one person to restrain him. The pain was incessant till near the time of his death. At 9.15 or 9.20 p.m. Dr. Little also saw him, to whom Percy complained of intense pain in the epigastrium, and of the skin feeling drawn up. He was retching. About 10 o'clock a quarter of a grain of morphia was administered hypodermically; and the symptoms abated, though not very much, about half an hour later. At 11 p.m., the severity of the pain having increased, one-sixth of a grain of morphia was injected; but it had no apparent effect in affording relief. At 11.10 he began to wander and became unconscious, his breathing became slower and sighing, and the heart's action weaker. Brandy did not revive him, and he died at 11.20, four hours and five minutes after the administration of the capsule, in which without doubt was enclosed the fatal dose—probably two grains—of aconitine.

The vomits were collected and mixed, some from a basin in the bedroom, some from the bath-room floor, and some from the pan of the water-closet, and were reserved for analysis.

The post-mortem examination made on December 6th, about 64 hours after death, showed the deceased, with the exception of the old spinal curvature and consequent atrophy of the lower limbs, to have had a well-built, muscular frame. There was no decomposition. The pupils were dilated. The membranes of the brain and the brain itself were slightly congested. There was no fluid in the ventricles nor any under the membranes of the brain. The lips were pale; the tongue pale, and as if bleached. There were some old pleuritic adhesions of the right lung. The lungs were healthy, but considerably congested, more especially at their bases. There was a small quantity of fluid in the pericardial sac. The heart was very flaccid, and as if stained with

blood pigment. All its cavities were almost empty. The liver, spleen, and kidneys were intensely hyperæmic. The mucous membrane of the stomach was congested throughout, and on the inner surface, near the cardiac end, there were six or eight small yellowish-grey patches, each about the size of a small bean, and a little raised above the surface. Towards the pyloric end there were two or three similar but smaller patches. There were three or four fluid ounces of dark fluid in the stomach, and of this a portion was reserved for analysis. The first portion of the duodenum was greatly congested, and there were patches of congestion in other parts of the small intestine. The bladder contained three or four fluid ounces of urine, which was also set aside for analysis. The membranes of the spinal cord were greatly congested.

The viscera of the youth were in the first instance handed to Dr. Dupré, of Westminster Hospital, who added rectified spirit to some of them. Next day, by order of the Home Secretary, they were handed to me for analysis, and at my request Dr. Dupré was associated with me in the analyses. I am greatly indebted to him for his co-operation and assistance. He was equally responsible with myself for the results.

The viscera received for analysis were as follows :

1. A portion of the liver, the spleen, and the kidneys, in one jar. To this half a pint of rectified spirit of wine was added on December 7th by Dr. Dupré, two days after the post-mortem examination.

2. The duodenum, and another portion of the small intestine, the cæcum and the colon, in one vessel ; these were treated on December 9th with rectified spirit of wine, when they were already somewhat green from decomposition.

3. The stomach, which was treated with rectified spirit of wine by Dr. Dupré on December 7th.

These viscera were separately dealt with (*i.e.* the contents of each jar separately) by a special modification of Stas's process, based upon the known fact of the readiness and facility with which aconitine undergoes a sort of modification, and splits up into decomposition products. The contents of each jar were repeatedly digested with rectified spirit of wine at ordinary temperature ; and they were then again thoroughly extracted with spirit made faintly acid with tartaric acid and warmed for

a few moments to 158° F. (70° C.). The two alcoholic extracts were then filtered and separately evaporated to dryness at a temperature which did not exceed 95° F. (35° C.). Each extract thus obtained was repeatedly exhausted with warm absolute alcohol, the solution filtered off and evaporated below 95° F. The residues thus obtained were exhausted with tepid water, filtered, and not till then were the extracts obtained in each case,—viz. the one obtained with unacidulated spirit, and the one obtained by means of acidulated spirit, mixed. The acid aqueous liquid thus obtained was in every instance repeatedly shaken with washed ether, and the ether removed by a pipette. The “acid ether” liquids thus obtained were evaporated and reserved for further examination.

After this exhaustion with ether the liquid was made alkaline by the addition of sodium carbonate, and then again shaken repeatedly, the first time with a mixture of one volume of washed chloroform and four volumes of washed ether (such a mixture floats on water), and then with ether; and by evaporation of these ethereal and chloroformic liquids an alkaloidal or “alkaline ether extract” was obtained in each case.

I may here remark that the use of a mixture of ether and chloroform in such proportions that the mixture floats on water, recently claimed by Mr. A. H. Allen as a novel modification of procedure in analysis for alkaloids, has been used by myself for years. The late Dr. A. S. Taylor was accustomed to employ a mixture of ether and chloroform for this purpose, and to vary the proportions of ether and chloroform, so as sometimes to obtain a mixture which would sink in water and at other times such a mixture as would float on water, according to circumstances; and he employed, I believe, such mixtures in lecture experiments.

These alkaloidal extracts were carefully dried, first at 95° F., and then *in vacuo* over sulphuric acid, and weighed. In the case of the liver a final and separate extraction was made with a mixture of acetic and ordinary ether, to remove any traces of morphia which might still remain in the alkaline solution.

The following were the weights of “alkaline ether” or *alkaloidal extracts* obtained:

Liver, spleen, and kidneys (two thirds of jar)	·093 grain.
Intestines (half of jar)	·170 „

Stomach (half of jar) 077 grain

Urine (= one and a half fluid ounces) . . . 077 „

Besides the viscera, the following articles were also analysed :

4. Four fluid ounces of urine. To this two fluid ounces of rectified spirit of wine were added on December 7th, one day after the withdrawal of the secretion from the bladder at the post-mortem examination.

5. About five ounces of semi-solid vomit, collected from the floor of the bath-room, the pan of the water-closet, and a basin in the bedroom on the night of Percy John's death. This vomit was mixed with five fluid ounces of rectified spirit of wine on December 7th, one day after the post-mortem examination.

The vomit was subjected to examination by nose and eye, with the aid of lens and microscope ; and it was found to contain the following substances :—lumps of fat, traces of striated muscular fibre (? boiled rabbit), sliced onion, starch (probably of wheat), dried currants, sliced candied fruit peel, apple pulp, essence of pine-apple.

Portions of the vomit and of the urine were treated by the same method as that employed in the viscera, and alkaloidal extracts were obtained. The urine, like the liver, was subjected to a final extraction with acetic ether, as the alkaloidal extract was found to contain traces of morphia. The alkaloidal extracts weighed—

From vomit ($\frac{3}{16}$ or approximately one ounce) 026 grain.

From urine ($\frac{3}{4}$ or three fluid ounces) . . . 077 „

All these alkaloidal extracts were tested (1) by taste ; (2) with phospho-molybdic acid for alkaloids, (3) for morphia with iodic acid and starch. (4) A portion was injected into the cellular tissue of the backs of mice, two experiments being made in each case. For the last-named physiological experiment the extracts from the liver, spleen and kidneys, were mixed with that from the stomach, as the taste of the two extracts was very feeble.

It was found that all the extracts had an effect on the tongue like that of Morson's aconitine ; that they all re-acted with phospho-molybdic acid like an alkaloid ; and that all of them were fatally poisonous to mice, the symptoms produced on these animals being precisely similar to those resulting from the simi-

lar administration of English aconitine. The effect of the extracts from the vomit and urine upon the tongue was very powerful and lasted six hours.

The further (acetic ether) extracts from the urine and liver re-acted for morphia with both the well-known sulpho-molybdic acid and iodic acid and starch tests.

These experiments convinced myself and Dr. Dupré, in a conclusive manner, that aconitine had been administered, and morphia also, to the deceased; that having regard to the quantity of aconitine in the urine, the dose had been a fatal one; and that the morphia had been administered after the aconitine. It may be added that the vomit was free from quinine; and that the hypothesis that a quinine pill had been the cause of death was negatived. This, as will be hereafter seen, was a matter of great importance in the case.

The "acid ethereal extracts" above referred to were diluted with weak spirit of wine, filtered, and the evaporated filtrates tested for digitalin, and bodies bearing a similar physiological action upon frogs' hearts (*vide* 'Guy's Hosp. Rep.,' 1866, p. 82), but they had no specific effects. It had been suggested to us that digitalin was the poison employed. Hence the importance of the above observation. It is proper, moreover, to remark that at the time of making the analysis only a few of the facts of the case were known to us. The sale of aconitine to Lamson in the Isle of Wight was not known to the police till their attention was drawn by us to the presence of aconitine in the quinine powders and pills; and I may add that it was Dr. Dupré who first drew my attention to the probable presence of aconitine in the powders.

It is unnecessary, in a medical publication, to do more than refer to the fact that a whole train of circumstances was brought to light, in no little degree suggested by the analyses, which showed conclusively that this was a skilfully planned, though in the end clumsily executed, murder, carried out with details of horrible atrocity; and the admissions of Lamson subsequent to his conviction, and prior to his execution, after an unsuccessful attempt to demonstrate his insanity, must have removed all doubt from the mind of any reasonable being of the prisoner's guilt. There is no reasonable doubt that Lamson, in the early part of 1881, endeavoured to kill his

brother-in-law by means of aconitine pills sent over from New York, and some of these fortunately fell subsequently into our possession at a time when Lamson might reasonably have expected that all trace of them would have been lost. This idea was by no means so clumsy as some writers on the case would have it supposed. To mix an alkaloid of little known composition and properties with another (quinine), the tests and properties of which are well known, is a procedure calculated to lead an unwary analyst into a trap. It was the striking physiological results produced upon myself by the barest tasting of one of those pills which drew my attention to the probable presence of aconitine in them. To administer a quinine pill, *plus* a fatal dose of aconitine, across the Atlantic, had Lamson's design then succeeded, does not appear to me a very clumsy conception. Perhaps it was not original; indeed, a very similar course of procedure is described by the brilliant French novelist Hector Malot, as having been employed, and successfully employed, by the murderess of the novel 'Doctor Claude,' to get rid of her rival. In the novel Madame Gillet substitutes a pill of her own manufacture, containing a fatal dose of onay, an arrow poison, and a vegetable cardiac poison, for one containing a medicinal dose of digitalin. Emptying the box of veritable digitalin pills into the palm of her hand, she places the fatal pill in the now empty box, and replaces the innocent pills. She then goes on a visit to a distant place until her unfortunate cousin has taken, after the lapse of some time, the fatal pill. In the novel the murderess not only succeeds in her design, but contrives to throw suspicion upon Doctor Claude, the husband of the victim of her crime; and he is ably but unsuccessfully defended by M. Vandam, the preparateur of Claude Bernard, who quotes the experiments "de Fagge et Stevenson et d'Homolle" (see 'Guy's Hosp. Rep.,' 1866, p. 37), in support of his views.¹

The second attempt, which we now know was made upon the youth's life, was of a similar but more daring nature, the

¹ I am indebted to a legal friend—who was himself actively engaged in the defence of Lamson—for directing my attention to the above-mentioned novel, published no long time before Lamson began his attempts upon John's life. The gentleman above referred to is of opinion that, with great probability, the perusal of 'Doctor Claude' suggested to Lamson the method of executing his

poison being purchased at no great distance from the place where it was used. The witness (Mr. Chapman) who gave evidence of the visit to Shanklin, I.W., was at first uncertain whether it was a pill or a powder which John said Lamson had given to him when they two were alone together. Eventually he came to the conclusion that it was a pill. The subsequent results of the analyses make it more probable that the aconitine was given in the vehicle of a quinine powder. But whether pill or powder was then given, there is no doubt that aconitine was administered on the eve of Lamson's departure for America. The attempt on life was in any case a singularly bold and reckless one; but if it had succeeded a chemist might easily have been misled if he had examined one only of the powders submitted to us. It was only by a careful examination of each individual powder that the fraudulent character of some of them was detected.

In the third and fatal attempt the scheme was altered. Presumably all traces of the previous attempts were obliterated. Doses of half a grain up to five sixths of a grain of the alkaloid had failed to produce death, and there is little doubt that a dose of two grains was now employed. To place this in the capsule was, to say the least, ingenious, for thus not only would the very peculiar local effect of aconitine upon the tongue be avoided, but time would be allowed for the escape of Lamson should suspicion be excited. Then there was the sugar, on which suspicion might be thus thrown. Fortunately it was the convict's own letter directing suspicion to the sugar which first excited suspicion as to the capsule being the vehicle of the poison. To give the whole of the poison purchased boldly in the presence of a witness, in full gas light, and so that no trace of it might remain anywhere except in the body of the unfortunate victim, under circumstances which, without exciting suspicion, might immediately take Lamson to the

crime. On mentioning these facts to a distinguished judge he told me that he had read the novel with the greatest interest, and that he had been recommended to peruse it by another eminent judge, as containing the best description of the French criminal judicial system ever published. The writer can speak in equally high terms of the description therein given of the procedure of the medical experts; the details are given with wonderful accuracy. There is no doubt that the *La Pommerais affaire* (1864) suggested to Hector Malot the plot of his brilliant story.

Continent, was a bold scheme. In Paris the wretched man found himself without means to go further, and with only just enough money to pay for his fare back, and his friends were not in a position to assist him with money. The police were on his track, and he returned to brave the matter out. He doubtless thought that aconitine was a substance so difficult of detection by chemical means that it would escape detection altogether. Fortunately conclusive evidence of its administration was obtained.

Aconitine, *aconitia*, or *aconitina*, is the chief vegetable alkaloid of *Aconitum napellus*, and various other species of aconite. It is, perhaps, the most formidable known poison, the minimum fatal dose being probably not greater than one twentieth grain for an adult. The term aconitine is, strictly speaking, applicable only to the alkaloid extracted from *Aconitum napellus*. A somewhat similar alkaloid, *pseudaconitine*, but not identical with the true aconitine, is extracted from *A. ferox*, or Indian aconite, or bishch. A third alkaloid, *japaconitine*, has been obtained from the roots of Japanese aconite, a drug which is occasionally an article of commerce in this country. The chemistry of aconitine, *pseudaconitine*, and *japaconitine*, has been investigated by Dr. Alder Wright, who assigns different but analogous formulæ to the three alkaloids. Physiologically I find that the three alkaloids named, produce upon myself similar effects; and that the symptoms produced by them when given to mice in fatal doses are undistinguishable. Prof. Thos. Fraser, however, informs me that he has detected slight differences in their physiological effects.

Aconitine is met with in commerce of various degrees of purity and activity. The cheap German, or "exotic" specimens, sold at twopence per grain, are often almost inert, and appear to consist chiefly of the products resulting from the splitting up of aconitine alkaloids under the influence of chemical reagents. *Pseudaconitine* and *japaconitine* are not articles of commerce. It is commonly asserted that the special preparation known as "Morson's aconitine," sold at from one shilling to eighteenpence per grain, is *pseudaconitine* prepared from the roots of Indian aconite (*A. ferox*). Mr. Morson assures me, however, that this statement is incorrect, and that the cultivated *A. napellus* is

alone used by him. His preparation is terribly potent; $\frac{1}{2000}$ th grain is always fatal, according to my experience, to mice, and the same quantity exerts an unmistakeable physiological effect upon my own tongue. Aconitine is usually amorphous or only obscurely crystalline; but Mr. Morson has furnished me with a specimen of his own manufacture in tolerably large hard crystals, of a pale amber colour. This specimen is so potent that $\frac{1}{3000}$ th grain, subcutaneously injected, speedily killed a mouse of twice the usual size of these animals. Dr. Thudicum has shown me a specimen of nitrate of aconitine made by M. Petit of Paris, in very white, and silky crystals (the English preparation is always fawn coloured), which he informed me was more potent than Morson's preparation. I regret that an application made by me to M. Petit for a supply has met with no response; and I regret this the more, as the sole fatal case of poisoning by aconitine prior to the death of Percy John, was due to the administration of Petit's preparation.

Besides the firm of Morson there are other makers of English aconitine of perhaps equal potency. Messrs. Hopkins and Williams supply a similar article, largely used in France. In the United States, where aconitine is also now largely used in medicine, I am informed that no aconitine is made, and that all is imported.

Cases of poisoning by aconitine have been rarely recorded, and I know of but two fatal cases, that of Dr. Carl Mayer in 1880, and that of Percy John, in 1881.

The first case, non-fatal, which I find recorded occurred in the practice of the late Dr. Golding Bird ('Lond. Med. Gaz.,' N. S., vi, 1848, p. 30). A scientific gentleman prescribed two and a half grains for himself in one dose. The purport of his taking it is not stated. He must have fallen, it was supposed, immediately, and struck his head against an article of furniture. There was speedy vomiting. Dr. Golding Bird did not see him till eight hours after the poison was taken. The patient was cold, collapsed, pallid, and bathed in perspiration. The heart's action was barely perceptible. The pupils acted to light. There was no paralysis whatever, either of sensation or motion, though this might have been present at an earlier stage. The intellect was unimpaired. The prominent symptoms were terrific vomiting—a kind of spasm, during which the patient turned

convulsively on to his abdomen, with intense contraction of the abdominal muscles, and jerked out the contents of the stomach with a loud shout produced by the spasmodic contraction of the diaphragm. This spasmodic action of the diaphragm is well seen when aconitine is injected into a mouse. In Dr. Golding Bird's patient this ejection of the stomach contents was repeated every minute or two. He could scarcely swallow, but the act was performed with a spasm like that observed in hydrophobia. After the lapse of fifteen hours the pulse became perceptible; but the spasm in the throat prevented him swallowing. The spasm and exhaustion continued till the end of twenty-four hours. In thirty hours the patient had recovered.

In the '*Lancet*' (1880, ii, p. 778) is a notice of the alarming results produced by the administration of pills of aconitine, containing each $\frac{1}{250}$ th grain of aconitine, four times a day. Cold skin and a cadaveric aspect appeared on the second day.

In 1880, three cases of poisoning by crystallised nitrate of aconitine occurred in Holland (Schmidt's '*Jahrbücher.*' 1881, No. 189, p. 122; '*Berl. Klin. Wochenschr.*,' June 14th, 1880, No. 24, p. 337), and one of them proved fatal. The first was the case of a weakly man, sixty-one years of age, suffering from chronic bronchitis and a febrile attack. For this there was prescribed a solution of nitrate of aconitine. The patient took five drops, containing $\cdot006$ of a grain of the nitrate at 7 p.m. This produced an astringent and burning taste in the mouth, extending to the stomach. At 9 p.m. the dose was increased to twenty drops ($=\cdot025$ of a grain); and this dose was repeated at 8 a.m., 11 a.m., 4 p.m., 9 p.m.; next day, at 10 p.m., a final dose of ten drops ($=\cdot012$ of a grain) was taken. In all one seventh of a grain of the nitrate was taken in seven doses. After every dose the patient was seriously indisposed, so that eventually his life was in jeopardy. The symptoms were a feeling of coldness, cold clammy perspiration, severe vomiting, difficult respiration, great lassitude, and the patient felt as if he were about to become paralysed. There were intermittent deafness and blindness, spasmodic twitchings of the whole body, but more especially of the muscles of the face. At one time he felt that he was dying, and stated that he had been poisoned.

The respiration became stertorous and quickened, then slow and gasping. There was no loss of consciousness. It is not stated that there was any loss of sensation, or any actual paralysis. He recovered.

In the second case, a man, æt. 62, took an undetermined dose of the same medicine. When seen he had cold, clammy perspiration, a weak, irregular, dicrotic pulse, and he was conscious. The respirations were short, laboured, irregular, and superficial. The pupils were contracted, and responded feebly to light. There was no difficulty in swallowing. There was great precordial anxiety, and *facies hippocratica*. Suddenly the pulse entirely ceased, though the cardiac beats could still be feebly heard; and a deathly pallor supervened. The patient rolled from side to side of the bed. The pupils were now dilated. Tonic convulsions of the facial muscles set in, with trismus; then three hours after the dose general clonic convulsion, and the patient lost consciousness. In five or six minutes muscular relaxation ensued, but the convulsion returned in a quarter of an hour. An hour later death appeared imminent. Vomiting now set in, the pulse improved, and in twenty-one hours the man was convalescent.

The third case terminated fatally. Dr. Carl Mayer, who had prescribed for the above patients, himself took from fifty to sixty drops of the solution of nitrate of aconitine prescribed for the first patient. This corresponds to one-thirteenth to one-twenty-first part of a grain of the nitrate. It may be assumed that the dose was probably one-sixteenth of a grain. The symptoms commenced in an hour and a half; but they were not accurately noted till 8 p.m., four hours after the alkaloid had been taken. He was then found with a small, weak, irregular, but not slowed pulse, cold skin, and contracted pupils. He had an astringent and burning pain in the mouth, extending to the stomach, and difficulty in swallowing. The tongue was swollen. There was great precordial anxiety. He complained of burning pain, weakness, and heaviness of the limbs, especially the legs, which felt cold. Suddenly vision was lost, and the pupils became dilated. Soon, however, they again contracted; and vision was restored. Vomiting was procured by tickling the fauces. At 4.40 severe convulsions first set in, with stertorous respiration, ringing in each ear alternately, and deaf-

ness. Ether was employed hypodermically, and its use was followed by renewed vomiting and convulsions. The pulse, nevertheless, improved, and ether was again injected. In a few minutes there was renewal of severe vomiting and convulsions, and the patient became unconscious; the pulse failed, and death ensued at 9 p.m., without return of consciousness, five hours after the administration of the fatal dose. On post-mortem examination the viscera were unusually charged with blood, and there was considerable hyperæmia of the stomach and small intestines, so that the colon and rectum appeared pale and bloodless by contrast. The intestines contained fæces, there having been no stool passed during the illness; and the bladder contained two and a half ounces of urine.

In these cases it was intended to give Friedländer's nitrate of aconitine, a weak German preparation. The dispenser used instead a crystallised preparation procured from Petit, in Paris. Plugge, to whom the analysis was referred, found that Petit's preparation was eight times more poisonous to animals than Merck's and 170 times stronger than Friedländer's nitrate. He failed to detect aconitine in a benzin extract of the viscera.

To these cases must be added that of Percy Malcolm John already related. His symptoms accorded with those exhibited in other cases where active aconitine has been administered.

A solitary case of poisoning by German aconitine (Merck's) is very crudely recorded. An analytical chemist took eight grains of aconitine after dinner with suicidal intent. Half an hour later the first violent symptoms appeared. A burning sensation in the mouth and throat first made itself felt, and this became more intense every minute; intense pains in the stomach ensued after thirty minutes, and these became so violent in a few seconds that the patient writhed, shrieking, with the most dreadful convulsions, and tried to strike the wall with his head. He was held with difficulty, and milk and oil were given. Very soon he became incapable of swallowing; he was seized with spasmodic cough, and wanted to vomit. In spite of emetics he could not vomit, however, till an hour after taking the poison, and then with great exertion a dark greenish fluid was ejected; but this afforded no relief to the pain in the stomach and the burning sensation in the throat, which

rendered swallowing difficult. The application of the stomach-pump afforded no relief. Exhaustion ensued after violent convulsions, and the symptoms reappeared with renewed force. At the beginning of the third hour the pains and convulsions attained such violence that death was expected every instant. In the fourth hour, after repeated injections of morphia, the patient seemed somewhat better. Previous to this he indicated that his skin was greatly irritated. This irritation of the skin, as of ants crawling, continued apparently the whole time, and whenever the intensity of the pains somewhat remitted, he scratched the skin of the face and breast in a convulsive manner till these were sore. His eyes glared wildly, sometimes resting with a fixed stare on one point. The convulsions were repeated at almost regular intervals, and the inclination to vomit continued, although vomiting did not continue after the second hour. At intervals of about forty minutes the patient seemed to lose consciousness, but only for a few minutes, and then the convulsions and the other symptoms reappeared with undiminished violence. Three hours after the onset of the symptoms he became incapable of intelligible utterance, but indicated that he felt giddiness; and soon after he appeared to lose sight. He threw himself wildly about on the couch, screamed, and uttered fearful groans. Exhaustion and apparent coma ensued, and then renewed attacks of the most violent description. Then difficulty of breathing set in, and he appeared to suffocate. At intervals he was conscious, indicated that he felt pain in the head and stomach, and was very thirsty. The pulse and body temperature fell considerably, and before death, which occurred at the end of twelve hours, exhaustion and unconsciousness set in, cold perspirations, and death-like pallor. Though death from asphyxia was all along expected, this occurred from syncope. The post-mortem appearances showed nothing unusual. The pupils were dilated, the interior of the mouth was pale, the brain and lungs were congested, the valves of the heart were very flaccid, the liver and kidneys were congested. There was inflammation of the stomach, and its mucous membrane was congested (*sic*). The alkaloid was found by chemical analysis in the contents of the stomach; but, very remarkably, none was found in the urine of the deceased. ('Med. Press,' May 24th, 1882, p. 439.)

Analysis.—Aconitine, pseudaconitine, and japaconitine—spoken of collectively as aconitine—are alkaloids exhibiting no precise chemical properties by which they may be distinguished from other alkaloids. They are precipitated by such general reagents for alkaloids as phospho-molybdic acid and the double iodide of mercury and potassium. When pure they afford no satisfactory colour reactions. Fortunately aconitine may be discriminated from other alkaloids by its peculiar physiological effects; firstly, its special action upon the nerves of sensation; secondly, its characteristic fatal effects upon animals.

The sensations produced on the tongue and adjacent parts by the three active aconite alkaloids, aconitine, pseudaconitine, and japaconitine, are precisely alike, and differ, in my experience, from the sensations produced on that organ by any other substance whatever; and I have tried seventy or more vegetable principles. The result of placing a drop of a solution of aconitine (containing say $\frac{1}{1000}$ th grain), or a particle of the solid substance, on the tongue, is at first a scarcely perceptible bitterness, and the taste is that of any ordinary alkaloid, and is transient. In about three minutes, however, and after the first sensation of bitterness has disappeared, an intense burning, and somewhat benumbing pain, is felt not only at the part to which the alkaloid was applied, but also radiating in all directions. Immediately after the commencement of this, moderate salivation comes on, with a peculiar feeling of dryness and constriction about the fauces. Although the alkaloid may not have come into direct contact with the lips, the burning, numbing feeling is marked in these, and more especially on the inner surface of the lower lip. These sensations continue generally for hours, with varying intervals according to the largeness of the dose, and are followed by a painful sensation of the part of the tongue to which the alkaloid was applied. This feeling is one of searedness, not unlike that produced by the application of a hot body to the part, so as just to stop short of producing a blister; but it is very persistent. When $\frac{1}{300}$ th grain of aconitine is employed the sensations described last from five to seven or even eight hours; and with even $\frac{1}{2000}$ th grain they may persist for two or three hours. These experiments must be made with great care. Before I became fully aware of the extreme potency

of minute doses of aconitine, and had heard of the case of Dr. Carl Mayer, I on one occasion placed the smallest quantity of solid aconitine which could be taken on the point of a pen-knife upon my tongue. At the most the dose did not exceed $\frac{1}{300}$ th to $\frac{1}{400}$ th grain. This was about half an hour after dinner. The local sensations were most intense—almost intolerable—and were still very unpleasant after the lapse of eight hours; but the more profound physiological activities of the poison were very decided. The irregularity of the cardiac beats was very marked. I was obliged to remain quietly seated, and avoid the least exertion. Even the act of turning in a chair sufficed to quicken in an instant the slowed pulse by twenty beats; and this susceptibility to exertion continued for at least seven hours. Next morning no unpleasant effects remained. The physiological or “tongue test” is an excellent and characteristic one.

Aconitine destroys mice with certainty, and after the exhibition of an unmistakeable course of symptoms, not easy to describe. Some years ago Dr. Fagge and myself made the observation (‘Guy’s Hosp. Rep.,’ 1866, p. 82) that frogs are not good animals on which to employ physiological tests for the detection of aconitine. In consequence, however, of the statements of Drs. Ringer and Murrell as to the extraordinary susceptibility of these animals to the effects of aconitine, Dr. Dupré and myself felt it necessary to investigate the subject anew. I ascertained that mice are equally susceptible with frogs, perhaps more so; and that the symptoms exhibited by the former when under the influence of aconitine are more marked than when frogs are employed. I therefore employed mice in my experiments in connection with the Lamson case.

I need quote only one of these experiments, made with a specimen of Morson’s crystallised aconitine, as an example of the potency of aconitine when given to mice. A very large mouse (244 grains) had $\frac{1}{3000}$ th grain of the alkaloid in extremely dilute aqueous solution of tartaric acid (just enough acid to dissolve the alkaloid) injected into the cellular tissue of the back. In ten minutes the animal retched violently, the retching being apparently due to an intense convulsive contraction of the diaphragm. Two minutes later the tail was quite insensitive to a prick with a needle, and the web of the foot—a very sensitive part—was also insensitive. The animal had

continued to retch violently, and was now partially paralysed as to motion in its hind limbs. Fifteen minutes after administration there was frothing at the mouth. Two minutes later the animal suddenly jerked itself on the side in violent convulsions, which continued till death eighteen minutes after the injection of the poison. Retching, convulsions, insensibility to external stimuli, and speedy death, are the chief visible effects of aconitine upon mice.

The alkaloidal extracts obtained from the viscera and vomit of Percy John, after being tested for the presence of an alkaloid or alkaloids, and yielding affirmative results, were tested by applying a minute fragment of them to the tongue. Affirmative results were obtained in every case. Morphia was also specifically sought for, and evidence of its presence obtained in the urine and in the liver. A portion of each extract thus obtained was then dissolved in water acidulated with a trace of tartaric acid, and injected beneath the skin of the back of a mouse. Comparative experiments were made on mice with pure aconitine. There was a striking similarity in the symptoms of all the animals thus operated on. As an example of the extraordinary delicacy of the tests, I may mention that the alkaloidal extract from the urine, which weighed $\frac{1}{3}$ th grain only, and represented $1\frac{1}{2}$ fl. oz. of urine, was tasted by three persons, and was tested for alkaloids and for morphia and strychnine specifically. The residue then sufficed to kill two mice with all the symptoms of aconitine poisoning. The taste of this extract was so intense that it persisted in one experiment five and a half hours.

I believe that hereafter there will be no insuperable difficulty in the detection of aconitine in forensic medicine.

LABORATORY NOTES
ON THE
WORKING OF THE HISTOLOGICAL CLASS.

By C. HILTON GOLDING-BIRD.

IN giving some account of the work done in the Guy's Histological Laboratory, in connection with the microscopical class, I wish to answer a question that is commonly put to me, not only by present but by past pupils; "What is the easiest way of getting sections of tissues for the microscope."

It is useless to refer such querists to manuals of practical histology, for in them they find a multitude of alternative processes, from which they do not know which to choose, even if the time necessary for a trial of the respective methods advocated were at their disposal.

And whilst there is no royal road to histological ends, as far as I know, yet it is quite possible to indicate a line of procedure that is both simple and at the same time amply sufficient for all purposes of the medical histologist, and by which he may attain the best results with the least expenditure of time and trouble. I formerly employed here many methods that I have now abandoned, both on account of the time that they demanded in the laboratory, and because I soon learnt that a multitude of methods did not simplify instruction.

This paper is, further, no condensed treatise on practical his-

tology, nor would this be the place for such an attempt; but whilst it registers a method that has worked well in this school for the last five years, it may well, I hope, serve as a guide to those who do not find it possible to make trial of each of the many methods that the text-books on the subject are bound to detail.

What I believe to be the best and shortest way of preparing tissues, cutting and staining sections, &c., is here indicated. It is applicable to pathological as well as to normal tissues, though for purposes of differentiation, more can perhaps be done by means of varieties of dyes in the former than in the latter case.

After describing the obtaining and preparation of the tissue, details will be given on the subject of cutting the sections, and especially of the method of ether freezing, which for ease, rapidity, and cleanliness stands first. When the manipulative part has been described, an abstract from the syllabus of the classes will be given, so far as the methods and choice of material for illustrating the different subjects are concerned.

The specimen.—The specimens for sections are mostly taken from the lower animals. The similarity of structure is on the whole sufficient for all class purposes; yet some must be taken from the human subject, as the retina and spinal cord. The objections to the human specimens are that they cannot generally be obtained soon enough after death; and unless death result from some sudden accident, the tissues are likely to be very altered in character. Thus epithelial cells which from an animal may be demonstrated clearly enough with their nuclei, may from man be indistinct from the granular and cloudy state of their protoplasm—the accompaniment of high temperature. It is in fact where epithelium forms the principal object in the specimen that the human subject is particularly unsuitable.

The size and shape of the piece of tissue or of the “block,” as it is shortly termed, must be such that it can be attacked simultaneously at all points by the hardening agent, and not so thick that it cannot readily be penetrated. Unless under special circumstances blocks should not be thicker than quarter to half an inch; and where the specimen will be afterwards cut by the freezing process, thin “slices” can take advantageously the place of the “block:” they will harden more perfectly, and can

often be prepared in a shorter time than that allowed for "blocks." Larger pieces, if desired, of a spongy tissue, as lung, can however be prepared.

Where the specimen will be embedded for hand cutting there must be *length*, at right angles to the surface that will be cut, for the embedding mixture to get firm hold: in a word, it must be a "block" and not a "slice."

The hardening process.—Those who want very large sections, and who mount everything in glycerine or Farrant's¹ solution, find Müller's fluid,² or bichromate of potash (1 p. c. solution) preferable, since it penetrates more quickly than chromic acid, which is always used here; for in normal minute anatomy at least, large surfaces present no compensatory advantage for the greater trouble they give in manipulation. Unless otherwise stated, it is therefore assumed that everything is hardened in $\frac{1}{4}$ p. c. (= one grain to one fluid ounce) aqueous solution of chromic acid.

This hardening is really tanning, and all histological specimens thus prepared are nothing but good leather; hence the longer action of weak chromic acid answers better than the shorter action of strong. The reason for having thin "blocks" or "slices" is now seen; for if they are thick, or if the acid be too strong, it may tan the outside and leave the inside to decompose.

What would altogether make a cubic inch of tissue is bulk enough for half a pint of the chromic acid solution; and this should be changed on the second, fourth, and seventh days, and then left alone till the end of another week. Ten to fourteen days is an average time for tanning specimens. The tissue must either be suspended in the fluid, which is best, or must lie on something porous, as wool; for if it lies on the bottom of the vessel it does not get attacked on all sides by the fluid, nor does the purer fluid up above reach it unless it be frequently stirred. Ordinary gallipots with sticks laid across their mouths, to which the specimens are suspended, are very convenient.

¹ "Farrant's solution consists of 'equal parts of gum arabic, glycerine, and saturated solution of arsenious acid.' Owing to its containing gum it becomes hard, so that the cover glass becomes fixed at its margin." (Rutherford.)

² Müller's fluid is an aqueous solution of bichromate of potash ($2\frac{1}{2}$ p. c.) and sulphate of soda (1 p. c.).

Another way of hardening, especially when it is not desirable to have many different vessels, is to have one large open one, such as a basin, full of the hardening agent. The tissue is attached by a thread to a small piece of cork; this floats on the surface and suspends the specimen an inch or two down. Thus the impure chromic acid falls away from the specimen to the bottom, and there is no need to stir the fluid at all. Also, since the thread is wholly immersed with the specimen it does not become rotten, as happens often where it passes from the fluid to a stick placed across the vessel. The corks are all numbered so that the specimens are known; they are also easily lifted out for inspection.

The same fluid that tans also *decalcifies* to some extent, for it dissolves out the salts of bone or tooth; but unless very small fragments or very thin slices of bone or tooth are being prepared (*e.g.* a cochlea), it is best to add nitric acid to the chromic-acid solution in proportion of 1 p. c. A large bulk of fluid should be employed, and the bone suspended in it. Thus, a cat's lower jaw with the teeth *in situ* is softened in about three weeks or a month.

A *saturated solution of picric acid*, to which more crystals of the acid are added from time to time, is useful for decalcifying where the soft elements (protoplasmic cells, &c.) are specially wanted, *e.g.* developing bone; but it is slow in its action, and as it does not tan like chromic acid, it is well to toughen the specimen for a few days in the latter, before coming to the next stage in the process of preparation.

Picric acid is used also as an ordinary hardening agent, but unless for some special purpose it is not recommended. It, however, interferes less with the staining process afterwards than chromic acid.

The toughening by the tanning process is transformed into hardening by putting the specimen now into spirit. But unless the spirit be constantly changed so as to get rid of the chromic acid as it dissolves out, the tissue will be over tanned during the spirit process and become friable. This may also be obviated by adding to the spirit one sixth of its bulk of glycerine. This reduces the chromic acid quickly to the sub-oxide (green), and stops its tanning action, and the spirit need not be changed.

Specimens for hand cutting are (when once turned quite

green in the glycerine and spirit) best transferred to pure spirit. But this is not requisite where freezing will be employed, though it is advisable if the specimen is to be kept for months or years.

The blocks may be labelled, where separate bottles cannot be used, with a strip of vegetable parchment tied to each and inscribed with a dark lead pencil. This will lie with the blocks for years unaltered in spirit.

Cutting the sections.—Three methods have been employed at various times in the laboratory, but the last is incomparably the best and quickest for class purposes, and, indeed, for private work as well. The time occupied in the various embedding plans is a serious item where one has other duties: whilst the sections obtained by them are not good without considerable practice.

Methods.—1. Embedding in wax. 2. Embedding in elder pith. 3. Freezing.

Where a large block has been hardened thoroughly, or where the specimen is very hard indeed, it is quite possible to get fairly thin sections without embedding. Thus, lens well hardened, or bone or tendon, need not be embedded, and for the class often are not; but the very best sections cannot thus be obtained. The *raison d'être* of embedding is that small surfaces teach as much as large (which are usually only repetitions of small ones), and therefore a rest is required for the razor to get an even section. Also the embedding substance has such a consistence as that of cheese, so that it cuts evenly, with a little resistance, and clings enough to the razor to give it steadiness.

1. *Embedding in wax.*—White wax or solid paraffin is melted with a third of its bulk of olive oil; when cool this is the embedding mass. The proportions vary with the season; less oil being added in warm weather; when perfect, the mass should cut readily, but not chip, on the one hand, nor begin to melt with the heat of handling on the other.

To embed, take a piece of the hardened tissue from spirit, and let its surface just dry: hold it on the point of a needle in the mixture (heated till just fluid and no more) for a moment, and then withdraw it with its coating of wax. This enables the wax to "bite" well. Into a small paper box or

pill box now pour some of the wax mixture, and so immerse the specimen, by transfixion with a needle, that when the block of cold wax is turned out of the mould the surface of the tissue to be cut lies in the centre of one of the surfaces of the wax. The needle can then be withdrawn.

Holding the mass in the left hand, and the razor moistened with spirit in the right, the sections are cut off *secundum artem*: they are freed from the wax by agitation in spirit.

There is a special method for spongy substances as lungs (*vide* lung). Good practice in section cutting can be obtained by cutting up a rather soft composite candle, the wick represents the specimen.

2. *Embedding in elder pith; Ranvier's microtome.*—A specimen may be merely held in the fingers of the left hand between two pieces of carrot or elder pith, and then cut as when wax is used; but this is a crude method.

Better, though hardly recommended (although most of the class sections were made thus for one session), is the use of pith in Ranvier's microtome. This instrument I have described elsewhere;¹ here I will only add that it is a brass tube, opening at one end into the centre of a smooth brass table, and having at the other a screw for pushing up the specimen in the tube to the level of the table, along which the razor is pushed. The specimen is packed in the tube with dry elder pith, it is then wetted and the pith swelling holds the tissue tightly. If the specimens have been well hardened the pressure does not hurt them; delicate specimens can be first protected with a coating of wax.

3. *Freezing.*—This is by far the best plan: it requires specimens to be less hardened and therefore less time is lost in preparing them; it is applicable to fresh as well as to preserved tissues, and in good microtomes the smallest and thinnest pieces of tissue (and the smallest are often the most valuable!) can be utilised. I have obtained a dozen good sections by its means by *splitting* up horizontally a piece of the human retina.

The microtome employed in this laboratory is the best yet invented, but I must just mention a very simple form of freezing microtome, within the reach of all, and answering admirably where the hand has had practice already in the use of the

¹ 'Quarterly Journal of Microscopical Science,' vol. xv, new series, 1874.

razor. It was introduced by Dr. Urban Pritchard some years ago.

It consists (I write from memory) of a solid cylinder of gun metal, with flat ends, four inches long by one and a half wide, with a handle fastened into one end (looking like the sponge holder of a battery). The metal is plunged into ice and salt, and when cooled to the temperature of this mixture is withdrawn, rapidly wiped, and thrust into a cylindrical jacket of thick felt. It is now grasped round this, and on the end of the metal, which just projects through the jacket, a small piece of the tissue is placed; it freezes at once, and is quickly cut up, whilst frozen, with the razor.

But specimens require a special preparation before freezing, whether in this or any other microtome.

Preparation of the specimen for freezing.—Fresh tissues do not require any preparation, but can be frozen at once; but they are of very little use for class purposes.

Hardened specimens must be thus treated. From the block cut a slice, the surface of which is to be sectionised, $\frac{1}{16}$ th of an inch thick, transfer it to water for a few hours to remove the spirit and glycerine, and then place it in gum mucilage for twenty-four hours at least. In this the specimen may remain for weeks until wanted, if a crystal of thymol is added to prevent mould.

The harder the tissue the longer it must be soaking. Prostate gland requires at least three days in gum, whilst hardened tendon, bone, and tooth are kept for weeks (some permanently) in gum and thymol. Ordinary tissue can be cut after twenty-four hours in the gum, but a longer time is better. Gum when frozen cuts like cheese or soft wax; and if the tissue is not permeated with it, but only with water, the ice may cause the would-be section to break into powder.

It is well to keep a crystal of thymol in the bottle of gum always; it does not interfere with the subsequent treatment of sections in any way. Chloral is not advised, it is a good preservative but prevents some methods of staining being employed: it also hardens.

The microtome.—The best freezing microtome is Williams',¹ and the freezing mixture is ice and salt; a modification of it

¹ Swift and Son, 81, Tottenham Court Road, W.C.

by Groves, of King's College, in which ether is employed, is better in many respects, and for home or intermittent work is cleanly, time-saving, and inexpensive to use.

Both forms are employed in this laboratory. When some hundreds of sections are being cut at a sitting, say enough for a month's work, the ice and salt form is often used; but during the past session the ether machine has superseded it.

Rutherford's microtome has had its day, and cannot be compared with Williams', which is now very universally adopted.

A. *The ice and salt microtome.*—It is a tub of hard wood, standing about six inches high, and closed at the top by a movable lid, also of wood, covered with a disc of plate glass. The centre of the lid and glass is perforated to allow of the projection of the circular, flat top of a column of brass, that "takes its origin" from the interior bottom of the tub. The tub being filled with ice and salt (4 parts of ice to 1 of salt) all round the brass central column, the lid is then replaced. In a couple of minutes the little brass table that projects through the lid has a temperature far below freezing, which is maintained as long as there is any ice unmelted: and this is from one to four or more hours, according to the heat of the laboratory. The tub has a hole at its side to drain off, by means of a flexible tube, the liquid formed in melting; but the machine lasts longer if this hole is corked up and the "liquor" retained.

A drop of gum placed in the little brass disc in the middle of the lid is at once frozen quite white and hard.

The razor is carried in a triangular brass frame, which stands on three legs (screws with ivory tips), and which slides about with but little or no friction on the glass lid, and as it does so, the edge of the razor slices off anything placed upon the brass table. In the apex of the triangle (equilateral) is the front leg or regulating screw, which has a very fine worm, and by turning it round the edge of the razor, which faces it, is raised or lowered. The angles at the base carry the remaining two legs by which the razor can be adjusted to the horizontal line. Combined movements of these screws with the front screw allows of the edge of the razor being placed at any angle.

To use the microtome, place a slice of the gummed tissue on the brass plate and so regulate the screws in the razor

frame, that the edge of the razor shall just catch the specimen, and at the same time be depressed one eighth of an inch or so below its back (*i.e.* the blade must not be quite horizontal), and that its point and heel shall be exactly on a level. A steady, rather rapid push now from right to left of the frame, planes off the top surface of the specimen, and by regulating the front screw, the sections can be obtained of any thickness, according to the toughness or the reverse of the specimens.

The action is exactly that of the carpenter's plane, and any one who knows how to adjust the blade of this tool, will at once know upon what plan to go with the razor.

If the specimen is fairly soft (*i.e.* has been long enough in gum) it is possible to "whip off" sections almost more quickly than one can count.

From the razor, as they accumulate, they are removed with brush or scalpel to a saucer of water, which dissolves out the gum.

It is now seen why a cut *slice* of a specimen need be hardened or at least "gummed" for this microtome, and the thinner the slice the more quickly it can be hardened, "gummed," and frozen. For the class, which consists of some ninety members, a piece of tissue one sixteenth of an inch thick will supply ample specimens, and often I cut the slices only about the one thirtieth of an inch thick, and thus ensure their freezing like cheese, from being penetrated thoroughly with gum.

As the brass table is very cold when the specimen is put upon it, it not unfrequently happens that the tissue will not stick to the metal. This is at times a great annoyance, but can thus be remedied.

Smear gum on the table, it instantly freezes, and perhaps will lift up as a slice of "cheese;" heat the blade of a spatula or glass slide, and put it on the gum so that it melts it on the table, it is compelled to freeze now more slowly and will "bite" well.

This preparation of a "gum table" is also necessary when a specimen has to be split up very accurately. Then it is best to cut off with the razor layer after layer of gum, till a smooth surface is reached, then freeze the specimen on that, and if the screws of the frame are not touched, the razor will be true for the flat specimen. In this way it is possible to split up the retina into many slices.

It is impossible to describe fully every hitch that may occur. I append, therefore, the principal difficulties that one meets with in beginning with this microtome, for there is something to learn in it, and it requires skill to use it, and that constitutes part of its charms.

Although the front screw is regularly turned, a section is not cut each time. This has cost me many hours' annoyance; it is due generally to one of the following causes:—The lid of the machine rocks from warping, or the asphalte has loosened from the glass plate, letting it move ever so little in the wooden lid, or the regulating screw works too loosely in the frame and shakes from side to side: or the back of the razor is so low that it touches the specimen as it passes over and so tilts up the edge; or dried gum on the glass lid prevents the triangle travelling evenly; or the specimen may not have "bitten" firmly into the brass table.

When a specimen cuts too hard, the gum has not soaked sufficiently; especially hard specimens to cut are transverse sections of tendon, bone, and tooth; they require sometimes weeks in gum. This difficulty may be temporarily surmounted by dipping the razor and frame in very hot water; the heat melts, whilst the edge cuts the tissue; also it is never best to attack the broad side of a specimen with the razor edge parallel to it: thus a triangular specimen should be cut from one angle.

The ether microtome.—Originally it was made on the model of the ice machine with a wooden tub. The accompanying woodcut gives its present pattern.

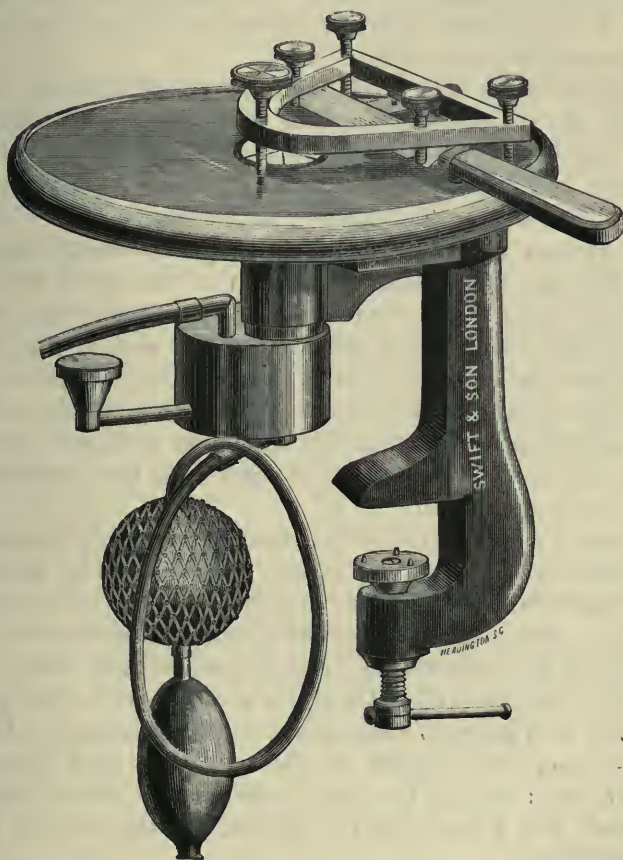
The wooden lid with plate-glass top are seen fastened to an iron leg, which is clamped to the table. The end of a hollow cylinder of vulcanite, held vertically in a collar projecting from the iron support, and capable of being adjusted or removed when necessary, is seen filling up the centre hole in the glass plate. The top of this cylinder is brass, and forms a table for the specimen, as did the top of the brass cylinder in the ice microtome.

In the interior of this cylinder is a "carbolic spray," which throws a jet of ether against the under side of the brass cap. The round box below the cylinder contains the ether, which is poured in through the funnel at the side. The tube bent at right angles carries off the spent ether, either into the room (if

it be a lofty one) or out of the window, whilst that to which the bellows are attached communicates with the jet inside.

The razor on its frame is also shown, but when being used the razor handle must be removed.

The bellows can be worked with the hand, or if a longer piece of tube is used, with the foot. In ordinary temperatures



thirty to forty seconds suffice for freezing for the first time, but in the summer 100 seconds are required. The ether employed is the methylated of sp. gr. $\cdot 720$; and two fluid ounces will last for three or four separate specimens.

Unless it is very warm weather there is no need to pump when once the tissue is frozen, it remains so quite long enough

to cut a hundred or more sections; where, however, the use of the microtome has to be *learnt* more ether must be at first expended. The greatest consumption of ether is when the instrument is being first cooled down at the commencement of a sitting. Also there is much waste in trying to freeze a thick slice of tissue; very thin ones should be therefore selected. As the freezing takes place after the gum is on the brass table, there is no difficulty about its adherence to the metal, as in the case of the ice machine.

So great is the cold produced that the moisture of the air pumped in, condenses as snow in the vulcanite chamber, and even may deposit on the jets and stop them up; but this last accident has been now prevented; it used in earlier instruments, however, not unfrequently to happen.

At present no means for condensing the spent ether has been devised, and except on account of the smell it is not worth trying after, for the cost of the ether is less than that of the ice and salt. Where the exit tube is carried out of the room there is no smell of ether at all. The manipulation of the specimen and of the razor is just the same in this as in the ice-and-salt microtome, and the difficulties to be overcome are much the same in both. So no more need here be said than has already been given.

Stains.—Aniline dyes are not recommended; they cannot be satisfactorily fixed, and only involve a waste of time. Carmine, too, though so long used, is rather capricious, for it will altogether refuse to stain a section sometimes (certainly when chloral has been used as a preservative) from no very evident reason. Its colour is rather dazzling for entire staining, whilst for double staining with logwood eosine takes its place very well. *Eosine*, *picro-eosine*, and *logwood* are the only dyes used here in a general way: and for the use of *gold chloride*, *nitrate of silver*, or *osmic acid* I must refer to cornea, endothelium, and nerve respectively.

Eosine is nearly of the colour of carmine; it is kept as a saturated aqueous solution; a few drops of this added to the water containing the section will impart a deep rose colour, but it may be used of any strength, for it is not capable of being fixed, so that in water the tint of the specimen can be reduced at once. When a section is soaked in the diluted

solution for some hours or even days enough remains to give a double stain with logwood.

For the class the custom is to put eosine into the bottles containing the sections a week before, then on the day they are required they are stained with logwood and mounted immediately after, for if left long in spirit after staining, they lose some more of the red colour; usually I put eosine also into the spirit in which the sections lie after the logwood staining and from which they are given out to the class.

Eosine is no good alone, its colour is too bright, and it is not selective; but with another dye, as logwood, it answers well, for the protoplasmic elements of a specimen are the last to lose their red eosine tint, so the logwood stains the older parts and the eosine the less differentiated. This is well seen in developing bone.

Picro-eosine is eosine solution saturated with picric acid, it forms a "cardinal" red as opposed to the rose red of eosine pure. It is used just as eosine, but it fixes to a certain extent; picric acid readily stains epidermal structures, and for these particularly is this dye useful. In studying hair follicles a transverse section of scalp stained with picro-eosine and then with logwood gives most instructive results. Picric acid does not interfere with logwood staining.

This double staining is not recommended merely from an æsthetic point of view, but in class demonstration, if one can say that one tissue is yellow, another blue, another red, the difficulties of the task are simplified.

Logwood solution is thus prepared: one ounce of the extract of logwood finely powdered, is mixed with fl. ℥vj of common hard water, and ℥vj of Liq. Potassæ, B.P., alum being added to saturation; a slight excess of undissolved alum is an advantage. The mixture must lie in an open vessel for six weeks to three months, constantly stirred, loss being made up with water from time to time. The process is one not only of solution but of oxidation, and hence cannot be hurried. When completed the solution should be a rich claret colour; and poured off from the sediment it will keep indefinitely provided a crystal of thymol is added. From the residue another though weaker solution may be made. Thus made it is too strong to use alone, unless staining instantaneously is

wanted, so it should be mixed with hard water, ten to twenty drops to the watch-glassful. It will now strike a bluish tint from the presence of the salts in the water, and that is why rain or distilled water is not to be preferred.

Since logwood is always "crusting in bottle" it must be filtered each time it is used. The few drops required are quickly run through a small filtering paper directly into the water.

If the logwood is used of the strength indicated, five to ten minutes will probably give the necessary tint to the section. The presence of any chromic acid prevents or delays the process, and if the specimen has been washed just before with slightly alkaline water it will stain the readier and with a bluer colour.

From the dye the specimen is rinsed in hard water, then transferred to spirit and mounted in balsam in the usual way. Those sections destined for glycerine are not transferred to spirit, but are mounted at once from water.

Preservation of sections.—Sections cut by embedding are freed from the wax in spirit, and those cut by freezing are cleared of the gum by floating in water for a few minutes.

Sometimes, and especially for the class, sections are made long before they are required for use. They can be preserved for two months in $\frac{1}{4}$ per cent. solution of bichromate of potash, and only require a few minutes soaking in water to prepare them for the staining; being thoroughly alkaline they stain very well. This is the plan most usually adopted here. For longer periods, Hamilton's fluid of glycerine and water, of each an ounce, and carbolic acid ten minims, is very good; but here I employ and prefer instead of the carbolic acid, a small crystal of thymol and two drops of *Liquor potassæ* to the two fluid ounces. Sections rather improve by keeping, for it ensures the complete removal of all chromic acid.

In private work the sections are separately handled with needles or flat metal lifters, but where a hundred or more have to be managed this is impossible. They are, therefore, passed on through the different stages by filtering on very fine cambric or muslin, and from the inverted filter they are rinsed off into the next fluid.

I think the specimens are less harmed in this way than by constant lifting; even retina or cochlea are thus treated.

In regard to *mounting* I need not add much. "Farrant's solution," already mentioned, is undoubtedly better than glycerine, but it is very hard to make so as to get the liquid quite clear and transparent; it is used sometimes in the laboratory, but it is not employed in the class.

The *balsam solution* is made by drying ordinary Canada balsam by heat and redissolving in methylated chloroform. It should have a density of very thin syrup. It also forms a good varnish for the glycerine specimens.

The *clearing medium*, through which the specimen must pass after being in spirit in order to be "miscible" with the balsam, is the carbohc acid and turpentine¹ solution. Many essential oils can be used, as oil of cloves, but they are longer in their action, and require that absolute alcohol should be used for the specimens, as with methylated spirit a cloudiness is likely to remain. After removing the specimen from the spirit it is floated on the surface of the clearing fluid in a watch-glass, and becomes transparent almost immediately; it then is transferred to the slide, the fluid drained off, and lastly mounted in the balsam.

The following sketch of the plan pursued here in the histology class will serve to illustrate the specimens chosen or the methods employed for teaching purposes, but which are equally useful for private study.

In the first part of the class, general histology is taken up, and in the second the special, which deals with the various organs. In the former, many specimens are not permanently preserved, and those that are, being often teased specimens, are mounted in glycerine. In the latter, sections are almost solely employed and are mounted in Canada balsam.

A. GENERAL HISTOLOGY.

Molecules, granules, Brownian movement (molecular movement), illustrated by gamboge rubbed up in water.

Simple forms of life, vital movement.—Bacteria and infusoria from hay infusion.

Protoplasm, its physical and vital properties.—The yeast

¹ Liquified carbohc acid, 1 pint; spirits of turpentine, 4 pints (Foster).

plant, leaf of the *Anacharis alsinastrum*, *Paramœceum aurelia*, frogs white blood-corpuscles (living), pus corpuscles.

By these specimens it can be shown that protoplasm is a transparent and homogeneous or more or less granular, semi-fluid, primitive, contractile, protein substance, either enclosed in a cell wall or free.

The animal and vegetable cell.—By means of the specimens just given and the further examination of a white blood-corpuscle, the following can be illustrated:—The word “cell” is a conventional term conveying a definite *physiological idea*, which applies equally to both animals and vegetables, and expressing a *physical state* which differs in the two kingdoms. *Physiologically*, the cell is the simplest body in which individualised life is to be found (nutrition, motion, reproduction); *physically*, it is a nucleated mass of protoplasm, in the animal kingdom; and in the vegetable kingdom it is the same within a cellulose wall.

Lymph and blood.—For *lymph* the cells of any serous effusion answer, or a scraping from a fresh lymphatic gland.

For *blood*, first frog's, then human blood under a cover glass without any reagent. In the former the amœboid white corpuscles are studied.¹ *Structure of the red* (zooid, œcoid) more quickly shown by irrigation with $\frac{3}{4}$ per cent. acetic acid, than the usual 2 per cent. boracic acid. The nucleus, when present, is demonstrated at the same time. *Hæmoglobin* is made from the whipped blood of a rat, in evaporating dish, with five times its bulk of water, and stood on ice for twenty-four hours. The crystals are deposited as a silky cloud. *Hæmin*, pure, from whipped and dried horses' blood, heated to 140° F., with a tenth its bulk of dried chloride of sodium, and ten times its bulk of glacial acetic acid, and filtered whilst hot; the crystals deposit in the filtrate on cooling.

The blood of various animals is then given. A smear dried on the glass slide will keep and show well. Frog's blood serves also as a micrometer ($\frac{1}{1000}$ in).

The *tissues* are aggregations of similarly altered and modified cells comprising the whole or part of an organism.

¹ To see the human white blood-corpuscles alive, a warm stage should be employed; a simple effective form of which will be found described in vol. xv, new ser., of the ‘Quarterly Journal of Microscopical Science.’

Epithelial tissue is studied from scrapings from the surfaces of the various mucous membranes that have been two days in 1 per cent. solution of bichromate of potash. They are examined in water. *Endothelium* is well shown on the omentum of a cat, that has been placed for thirty minutes in $\frac{1}{2}$ per cent. solution of nitrate of silver; it has then been exposed to light in glycerine and water until dark brown. A thin fragment may be permanently mounted in glycerine. If tinted with logwood the omentum shows also connective-tissue corpuscles, white fibrous tissue, and adipose tissue. *Cilia and ciliated epithelium* are seen dead in the cells from the tracheal mucous membrane, and alive from the gills of the mussel, examined in salt and water to show their characteristic movement.

The fresh tendon pulled out of a rat's tail shows *white fibrous tissue* if teased up in normal salt solution¹: it is seen also in the fine bands of the omentum, especially when stained with logwood. The fresh specimen irrigated with acetic acid shows the "tendon-cells" or connective-tissue corpuscles.

The corneal corpuscles are here given in further illustration of this subject (see "Eye").

The *fibrils of white fibrous tissue* are well seen by teasing in glycerine a piece of fresh tendon that has soaked a few days in a purple solution of permanganate of potash.

Sections of hardened tendon, transverse and longitudinal, mounted in glycerine form permanent specimens.

Yellow elastic tissue is given from the lig. nuchæ of the ox, teased in glycerine, after maceration in spirit and water acidulated with acetic acid.

The *development of connective tissue* is illustrated by a section through the wrist-joint of a fœtus about the sixth month. There can be seen developing white fibrous tissue from the embryonal corpuscles, as well as its relation to cartilage. This specimen is most instructive as giving ocular demonstration of the transformation of the fœtal elementary protoplasmic cell, the direct lineal descendant of the ovum, by division into a higher and permanent tissue (see also "Blastoderm"). It has an important bearing upon the histology and physiology of repair of wounds.

The *indirect method of development of white fibrous tissue*, as

¹ .5 to .75 per cent. chloride of sodium in water.

shown in Wharton's jelly, is best seen, if the cord be cut fresh by freezing, and examined in iodine water; but specimens hardened in chromic acid and stained in the usual way are best for class purposes.

Adipose tissue seen in the omentum. Its relation to connective tissue shown by examining freshly teased gelatinous tissue from floor of rabbit's orbit.

For *neuroglia* see Spinal cord.

For *adenoid (connective) tissue* see Lymphatic gland.

Connective-tissue group.

All the various forms of *cartilage*, stained with logwood, mounted in balsam. One section only, fresh from frog, to show unchanged protoplasmic cells. Simple cartilage seen in embryo chick (notochord), and in sections from a mouse's ear.

Relation of cartilage to connective (fibrous) tissue see vertical section through foetal spinal column.

Bone (decalcified) shown from human subject, in various sections stained with logwood and mounted in balsam. Marrow (red) from cats' ribs, fresh, teased in normal salt solution.

Development of bone.

In membrane, shown to be merely "periosteal" by sections of two adjacent layers of periosteum where commencing to make bone, as in foetal skull, through a suture, at seventh month.

In cartilage, various sections through bones of kitten at birth, and especially vertical sections through bodies of vertebrae, where, besides developing bone, the development of fibrous out of hyaline cartilage, and the relationship of connective tissue to these can be studied. Also vertical sections through ends of long bones and transverse ones through their diaphyses.

Preparation (from the new-born kitten).—The whole animal being skinned and disembowelled, can be put into two quarts of saturated picric acid, with an excess of the crystals, until decalcification is complete, and then be kept in spirit.

All these sections should be stained with eosine and logwood and mounted in balsam. The foetal protoplasmic elements and the blood are specially shown by the eosine, which aids materially in demonstrating the source of the periosteal ingrowths, and the fact that none of the cartilage makes permanent bone.

Tooth. Its development.

The tooth *in situ* is shown well by vertical sections through an adult cat's lower jaw.

Preparation.—With a fine saw slice up the jaw in pieces one eighth of an inch thick, and then soften in 1 per cent. nitric acid, and when soft, keep a week in $\frac{1}{4}$ per cent. chromic acid solution. This specimen takes long to prepare, and requires also long soaking in gum, if cut by freezing.

A transverse section through the fang of a sheep's tooth, similarly decalcified, shows dentine, pulp, and crusta petrosa, &c. The *enamel* is demonstrated by soaking sheeps' teeth in 1 per cent. nitric acid for twelve to eighteen hours; the enamel can then be cut off like chalk and broken up with needles, in glycerine, to show the prisms. The lower jaw of the kitten, decalcified as directed before, shows well, in vertical section, developing tooth with enamel, dentine &c.

All these specimens are stained best with eosine and logwood and mounted in balsam (enamel excepted).

Muscle.

Non-striated muscle is shown by hardening cat's intestine in 1 per cent. nitric acid solution and then in 1 per cent. bichromate of potash for fifteen days. It is kept in glycerine. Thin strips of muscle can be easily peeled off and mounted in glycerine, after teasing with needles.

Heart muscle is similarly treated; sections of heart are shown also.

Striated muscle in section is best shown in tongue, one section giving fibres in all directions. *Muscle from the skate,*

prepared as the intestine above, and then pounded in a mortar with glycerine, shows admirably sarcoous elements and fibrils. The drop of glycerine containing the pulverised muscle is at once to be mounted as a permanent specimen.

A transverse section of the tail of a young rat prepared in chromic acid is here given as illustrating much of what has already been taught in the class.

Blood-vessels.—The smallest vessels are demonstrated by examining the pia mater (the fringes that lie between the convolutions especially) of sheep after lying three days in 1 per cent. bichromate of potash. It is then rinsed with water and tinted with logwood.

To see the endothelium of the vessels a pia mater must be injected with nitrate of silver, but this is not given as a class specimen.

The larger vessels are shown by sections stained with eosine and logwood. They must be cut very thin, and should be as deeply stained with the eosine as possible. The renal artery is a good typical one.

Nerve-fibres (medullated or white).

Fibres from the lumbar plexus of a frog placed for twenty-four hours in 1 per cent. solution of osmic acid, are split up longitudinally with needles and examined in glycerine.

Sections of nerves in bundles (*e.g.* ulnar), and of superior cervical ganglion of the sympathetic, are mounted in balsam, after picro-eosine and logwood.

Non-medullated or grey nerves are studied in the spinal cord and in retina.

Nerve endings, as Meissner's corpuscles and Paccinean bodies, are seen in the sections of pulp of finger or toe.

B. SPECIAL HISTOLOGY.

This includes sections of skin, mucous membrane, and all the organs of the body; so only those that are specially prepared or specially instructive will be indicated here.

Section through lip of fœtus at full term is instructive for demonstrating the transition between the skin and mucous membrane.

Skin over tibialis anticus, or section of a papilloma, shows "cogwheel" epithelium *in situ*.

Pulp of finger is given as typical of skin, and to show "end organs" of nerves (*vide supra*).

Transverse sections of scalp at different levels demonstrate the structure of the hair root and follicle; they are best strongly stained first in picro-eosine and then tinted with logwood.

Small intestine of fœtus at sixth month shows villi in an exaggerated degree; and proves them to be only leaf-like prolongations of the septum between two Lieberkühns follicles.

Spleen of rat is a good class specimen, as the Malpighian bodies are well-marked; this specimen and that of *lymphatic gland* (of sheep) require to be very thin and deeply stained, first with eosine and then with logwood.

Kidney is best shown by entire cross vertical sections from the gland of a rat or guinea-pig, supplemented by a section across the tip of one of the papillæ.

Lung.—Where this is to be cut by freezing, the mode of preparation used for other things is employed, only that the hardening fluid wants to be also injected into it through the trachea. The final hardening is in spirit. Where it is to be cut by hand, the hardened tissue is soaked well in gum and then plunged into spirit; this coagulates the gum in the air-cells and renders the specimen quite hard. It then is embedded. If the sections are floated into water the hardened gum at once dissolves out.

Ovary and ova are best shown in sections of the ovary of young animals (as bitch). The formation of the Graafian follicles and of the ova can be then studied. The fully formed follicle, however, is best seen in sections from the adult. Each section should include the end of one or other of the long diameters of the organ.

Prostate.—The post urethral part, *i.e.* under the floor of the prostatic urethra, is the part to be used, as it contains the glandular elements.

Special senses.

Taste bulbs are seen in vertical sections of the plaques on the sides of the rabbit's tongue; they must be at right angles to the line of the furrows in the mucous membrane, and be cut very thin.

The *olfactory mucous* membrane is studied from the hardened and decalcified upper spongy bone of the sheep; or by cross sections through the head of a newt that has been prepared by the picric acid process.

The *cochlea* is best taken from the guinea-pig; the large tympanic bulla, just behind the articulation of the jaw, is opened with scissors, and the cochlea is seen standing out freely from the inner wall; it is excised and suspended in picric acid solution, and when decalcified is placed for five days in $\frac{1}{6}$ per cent. chromic acid, afterwards in spirit and glycerine. Before placing in gum, a vertical slice close to the margin is taken off parallel with the long axis so as to open the tube and admit the gum. By the freezing microtome forty to fifty serviceable class specimens may be made from one guinea-pig.

The *retina* must be human and obtained not from the post-mortem room but from an excised eyeball, where the front part only is diseased. It is hardened as other specimens, but the eyeball is first cut in two with sharp scissors $\frac{1}{4}$ inch behind the cornea. The vitreous is then turned out under the surface of water, and the cup, or posterior part of the ball, is now lifted up full of water. It is rapidly transferred to $\frac{1}{4}$ per cent. chromic acid without emptying out the water. The process is completed just as in other specimens. Thus prepared, pieces of retina $\frac{3}{16}$ in. square are excised with the choroid attached, and after passing through the various stages are cut up by the freezing microtome. If the brass table is already at freezing point, the square fragment is easily stood up on its edge, supported against and embedded in a drop of frozen gum.

Sections through the yellow spot are also given to shew the peculiarities of the outer nuclear layer.

The retina, prepared as described, usually preserves the outer

element of Jacob's membrane, and the pigment layer *in situ*, even where the choroid becomes detached.

Instructive *meridional sections*, including junctions of cornea and sclerotic, of iris, choroid, and ciliary muscle, and the front of the retina, can be obtained from the eye of the sheep: the back of the eyeball of a smaller animal, or of man is used for sections through the *optic disc*, especially to show how the nerve-fibres come to lie inside the retina.

The *corneal corpuscles* are most uncertain in their preparation. For class purposes the following plan answers best. The cornea of a sheep, excised within fifteen minutes of death, has its anterior epithelium roughly scraped off with a sharp scalpel, and its substance is scored across in several places. It then is immersed for two hours in $\frac{1}{2}$ per cent. solution of chloride of gold in the dark and then transferred to distilled water acidulated with acetic acid; it is exposed to direct sunlight and warmth, till dark purple. It is then cut up into small pieces ($\frac{1}{4}$ in. across) and also split once with a sharp knife, and transferred to gum. Being a dense substance the gum may take some time to penetrate, but the light can be acting on the specimen all the while; the cornea cannot become too black. It is then split up by the freezing microtome into the finest sections, which in a good cornea should be blue or blue red. If the corpuscles do not now show well the sections may be further exposed in formic acid and water to sunlight. When the change is completed they may be kept any time in glycerine, in the dark.

By this means I have obtained the corpuscles and nerves in the same specimen, but there is no certainty as to how the specimen will turn out, and it cannot be prepared save in the summer; it then takes about three weeks.

It is important that the sheep's cornea and not that of the cat should be used; the corpuscles are better seen in the former.

Development.

The living chick's blastoderm is shown to illustrate the early changes in the ovum.

The blastoderm up to the fifth day is easily manageable, and

can be kept alive some time in warm water. The chick on a later day soon dies, but up to a week, though not easily shown alive a better idea of the amnion sac, of the yolk sac, and of the great extent of the blastoderm can be gained.

The eggs are incubated in the laboratory over gas, after the method explained in Balfour and Foster's 'Embryology.'

To exhibit them to the class they are broken into water at 40° C. and then the embryo can be demonstrated *in situ*. At sixty hours, the area pellucida and area opaca, the vascular area, and the circular sinus, are well seen, though the actual extent of the blastoderm is not well observed unless the yolk is manipulated with forceps. The chick lying on its side, the bent tubular heart, pulsation can be readily distinguished with the naked eye.

To prove the tangibility of the blastoderm, one is cut from the yolk, beyond the circular blood virus, and removed as a thin lamina with the embryo dependent in the middle; it is now floated into a cell made of thin cork fastened to a piece of glass 6 × 4 in., and covered with another smaller plate of glass. The whole operation is carried on under warm water in the presence of the class. The plate on which the cell is made has also a cork glued to each of its corners, so that it floats about an inch below the surface of the warm water in a horizontal position. Thus the vessel of water can be handed round without fear of disturbing or folding up the blastoderm.

A section through the body of an embryo of a chick on the third day is usually given to prove that at an early age the animal is a mere collection of cells derived from the ovum by division, as well as to show the notochord, formation of neural canal, &c.

Lastly, embryos in the blastoderms already mounted as transparent objects are shown under low objectives, whereby the formation of the amnion is well seen, growing as a hood over the embryo from the head downwards.

The bent tube forming the embryo heart is thus seen as well.

The idea constantly kept before the class is the prime importance of the protoplasmic cell from which everything else comes; the class both opens and closes with its study, though at opposite ends of the life cycle.

CASES
OF
PARALYSIS OF THE ABDUCTORS OF
THE VOCAL CORDS.

By FREDERICK TAYLOR, M.D.

IN this paper I propose to record some cases of laryngeal paralysis that have come under my notice at Guy's Hospital during the last two or three years. Many points with reference to this subject still remain open for discussion, and it is hoped that the cases may form a contribution having a certain amount of interest.

It is unnecessary here to enumerate all the separate varieties of paralysis of which the laryngeal muscles may be subject; we know that the paralysis may be unilateral or bilateral, and that in bilateral cases a broad distinction may be drawn between cases in which only the abductors or glottis-openers are paralysed, causing an obstruction to inspiration, and those in which only the adductors or glottis-closers are affected, with the result of producing aphonia.

Further, experience has shown that while the latter cases are always functional (hysterical aphonia), the former are nearly always organic, the result of pressure on the recurrent laryngeal nerve or of some lesion presumably affecting the nerve-

nuclei; and their clinical features, as already indicated, are widely different, the one being characterised by the impossibility of approaching the vocal cords to one another, so that their vibration of, and by, the expired air cannot be effected, and hence aphonia; and the other resulting in a permanent median position of the cords, with a narrow chink through which the air is forcibly sucked with loud stridor, while phonation is quite or nearly perfect. But all the muscles of the larynx, except the crico-thyroid muscle, are supplied by the recurrent laryngeal nerve, and it would be expected that any lesion of this nerve or of its nucleus would paralyse alike the abductor and adductor muscles. When the solution of continuity is complete, as in section, this, of course, occurs; and as a result of pressure by growths in the neck or chest, both groups of muscles may in the end be affected. But it remains one of the most interesting facts in the history of laryngeal paralysis, the explanation of which is still wanting, that in injury or disease of the roots or trunks of the pneumogastric, spinal accessory, or recurrent nerves, the abductor filaments are liable to become affected sooner than the others, and may be affected alone. This fact is now accepted by laryngologists, and was more or less considered at the International Medical Congress, 1881, in the papers of Professor Gerhardt, of Würzburg, and Professor Lefferts, of New York, and by Drs. Rosenbach, Semon, Bosworth, and Burow in the discussion which followed. In the 'Archives of Laryngology,'¹ Dr. Semon strongly insists on the early implication of the abductors, whether disease affect the centre or the trunk of the laryngeal nerves; while, on the other hand, the adductors are never similarly picked out in organic disease, though, as already stated, they are peculiarly liable to functional failure. No one of the explanations that have been suggested in the case of the abductor proclivity is entirely satisfactory. The hypothesis of the existence of an independent ganglionic centre for the abductors leaves open the question why it is so often affected and the other centre so rarely; while no proof is forthcoming to support the suggestion that there is a special arrangement of fibres in the recurrent laryngeal nerves, the

¹ Vol. ii, No. 3, July, 1881.

adductor fibres being central and protected, and the abductor filaments peripheric and exposed.

The additional nerve force that the adductors receive through the superior laryngeal nerve must be slight, and is insufficient to explain the difference observed in some cases; while to say that the abductor fibres possess a specific vulnerability is not much more than to re-state the facts.

The case which follows is an illustration of the condition above referred to, and presents features of interest which will be alluded to afterwards.

CASE 1.—*Paralysis of abductors of vocal cords; cancer of the œsophagus, involving the recurrent laryngeal nerves; tracheotomy; broncho-pneumonia and gangrene of lungs; atrophy of several laryngeal muscles. Death.*

(Reported by Mr. H. BLATHERWICK).

Daniel D—, æt. 58, was admitted under my care into Guy's Hospital, on January 22nd, 1881, and gave the following history. He was a foreman of labourers, and much exposed to wet and cold. He is married and has had three children.

Family history.—His father and mother both died about the age of sixty, the father of rheumatism and cold; the mother had ulcers on the leg. His only sister died in childbirth.

Previous history.—Forty years ago he strained his neck while carrying a plank, and he was in the hospital and was cupped just below the neck. In March, 1880, he was struck on the middle of the sternum by a piece of timber weighing about three tons, and was forced at the same time against the wall. He was laid up in consequence for six weeks, and then going to work continued to have a pain in the back for two months more.

His present illness commenced six months ago (July, 1880), when he first noticed that food stuck in his throat, and ever since then he has had increasing difficulty in swallowing, so that he is a long time getting his food down. Nine weeks ago he first observed a whooping noise during respiration, especially when he exerted himself, and for some time previous to this, there had been difficulty in breathing.

On Saturday, January 22nd, he presented himself among Mr. Jacobson's out-patients complaining of the dysphagia. Mr. Jacobson tried to pass an œsophageal bougie but failed, and seeing that there was a laryngeal complication, sent the patient into a medical ward.

Present condition.—He is a well nourished man, but states that he has lost one and a half stones in weight during the last nine months. His face is dark from exposure, the hair getting grey, and there is well-marked arcus senilis in both eyes. His most obvious difficulty is that affecting his breathing, which is stridulous during inspiration. The stridor is scarcely audible during quiet respiration, but on any exertion it becomes well marked, and is brought out distinctly as a loud whooping noise during forced inspiration. During sleep this noise occurs with every inspiration, and so loud is it, that all the other patients in the ward are disturbed. The chest is well formed, but in quiet breathing the movement is almost entirely diaphragmatic, and on forced inspiration it moves but very little more.

At the right apex posteriorly there is slight dulness with prolongation of the expiratory murmur. Elsewhere, both back and front, the resonance is good, and vocal fremitus normal, but the vesicular murmur is feeble and masked by the laryngeal stridor, and there is some wheezing with the expiratory murmur. He speaks loudly and distinctly, but somewhat as if he had something in his mouth.

The heart is normal in position and extent of dulness. The sounds are not very distinct, but there is no murmur. The right pulse is very slightly less full than the left, but it has a good percussion wave, and otherwise feels normal, nor is there any physical sign of aneurism in the chest. The other viscera appear healthy, hepatic and splenic dulness being normal, and the urine of sp. gr. 1011, with slight excess of phosphates, but no albumen or sugar. The tongue is slightly furred, the bowels regular, and his appetite is good. But he has great difficulty in swallowing, and is unable to take any solid food. With fluid he can take only one or two mouthfuls at a time. If he tries to drink continuously he chokes, has spasmodic closure of the glottis, with apnœa and cyanosis; and returns the fluid partly through the nose.

On the 25th I examined the larynx with the mirror and

found complete paralysis of the abductors of the vocal cords. The cords lay so close together that only the smallest chink existed between them, and there was only the slightest alteration with the respiratory movements. The aperture was median in position, and of the same width, about $\frac{1}{20}$ th inch, from front to back of the glottis. At the same time I attempted to pass a bougie, but failed. On the first occasion it seemed to go down about twelve inches from the teeth, but subsequently I could never get it farther than seven or eight inches. Examining the neck for tumour between the trachea and œsophagus, I could find nothing.

The diagnosis which seemed most probable then ultimately proved to be the correct one, namely, that he had œsophageal cancer involving the recurrent laryngeal nerves, and causing paralysis of both vocal cords.

He was ordered to take twenty-five grains of iodide of potassium three times a day, and to have a subcutaneous injection of $\frac{1}{30}$ th grain of sulphate of strychnia every night.

On the 27th Mr. Golding-Bird saw him with me with a view to having tracheotomy performed, as the dyspnœa was becoming daily worse. He also tried to effect a passage into the stomach, beginning with an olivary bougie, and trying soft gum-elastic instruments afterwards; but he did not succeed in getting them farther than eight inches from the teeth.

January 28th.—Tracheotomy was performed at 1.30 p.m. without chloroform. A large silver tube was inserted, and the patient was placed in a steam-tent by the fire. The tracheal cartilages were ossified.

Considerable relief was afforded by the operation, and even his dysphagia was after a time improved. But he was troubled from the first day after the operation by the return of small portions of his food through the tracheotomy tube; an occurrence which gave rise to the suspicion that there was a communication by cancerous ulceration between the œsophagus and trachea. No such aperture could be seen when on February 4th a strong light was thrown by means of a mirror on to the posterior tracheal wall through the operation wound; and ultimately it was shown that the food ejected must have passed first through the glottis. In spite of this, however, his swallowing was so much better that he could take solid food in

small quantities very soon after the operation, and got up within eight days (February 5th). On February 11th he could talk aloud when he closed the orifice of the tube. He was troubled with obstinate constipation. Auscultation of the left base showed deficient entry of air with some crepitation. The improvement, however, was only temporary, the dysphagia again became worse, the symptoms of pneumonia supervened, and he died on May 2nd, in the tenth month of his illness, and three months after the operation. The following short notes will show the progress of the case:

February 19th.—Feels stronger every day. Can drink two or three mouthfuls of fluid continuously. No food returned through the tube given the 10th inst.

22nd.—He can himself change the tracheotomy tube. He coughs much mucus through the tube. He has now left the steam tent to sleep in the open ward.

25th.—A little food and much mucus expectorated. Some wheezing rhonchi heard at the right apex in front.

March 3rd.—Dyspnœa, and increasing dysphagia; much food coming through the tube. Takes milk only. Has been again placed in the steam tent.

10th.—Has again had solid food since the 6th.

13th.—Fluids return through the tube; cannot swallow solids.

24th.—Has taken solids well since the 16th.

During the next month he continued much the same. The ability to swallow varying every two or three days.

April 24th.—Dulness at the left base.

25th.—Has taken twenty ounces of milk during the day.

26th.—The milk given during the day having been returned through the tube, he was ordered nutrient enemata.

27th.—Nutrient enemata continued, as much of what is given by the mouth returns.

29th.—Dulness, tubular breathing, and muco-crepitant râles at the left base behind and in axilla.

The temperature taken at intervals during the last nine days of life showed little that was typical. It varied from 99° to 103·8°, being often highest in the morning.

Dr. Goodhart made the post-mortem examination ten hours after death. A large tough mass, still lobulated in structure,

and infiltrating the neighbouring tissues, was found on each side of the neck behind the sternal end of the clavicle. This was hidden deep down in the root of the neck. The recurrent laryngeal nerve was caught on both sides and adherent to the growth; on the right side it was so adherent and so incorporated with the growth that it was impossible to detach it.

The larynx looked healthy. The vocal cords were closely approximated at their hinder third; in the anterior two thirds they were separated, having concave margins and forming a fusiform aperture. The posterior crico-arytænoid muscle was extremely wasted on each side; in place of a plump belly of muscle, it was reduced to a very thin band. The aryttænoides muscle was pale and flabby, and both this muscle and the crico-aryttænoides lateralis were very much wasted. The cricothyroid muscle also appeared flabby.

The œsophagus was tightly strictured in two places; one an inch and a third below the cricoid cartilage, and the other one and a half inches below this. The upper stricture was due to a soft mass of cancer which projected into the calibre of the œsophagus, and the other was like it. Between the two the mucous membrane was for the most part healthy, but in the hinder wall was a strip of cancer of rather nodular surface running between the two strictures above and below, and in addition there were two or three small circular flattened nodules in the mucous and submucous tissues, and Dr. Goodhart thought it not unlikely that the upper stricture had formed from the lower or *vice versâ*. They appeared to be about the same date; neither was much ulcerated on the surface. The glands in the neck were involved opposite the upper stricture, *i.e.* one and a half inch below the lower border of the cricoid. The trachea was not involved, except that there was one small white nodule just showing through its posterior wall.

Microscopically the growth was a good specimen of cancer; most of the cells being angular and epithelial.

The lungs presented much broncho-pneumonia in all parts; each lower lobe was collapsed, and there was a patch of gangrene at the lower part of the left upper lobe. There was also recent pleurisy at each base. The heart was healthy, but the base of the aorta was slightly atheromatous. The liver

showed near the surface a minute granule, consisting of fibrous tissue enclosing numbers of sections of tubules; Dr. Goodhart was in doubt whether this was an adenoma of the liver or a local cirrhosis which had caught some of the ducts, or made atrophied liver-cells look like ducts.

This case may be at once classed with those above considered, and agrees with them in the existence of destructive pressure upon the nerves supplying the larynx, in the atrophy of the muscles thereby caused, and in the marked failure of the abductor muscles before the adductors. It is necessary to draw particular attention to this because it is seen from the post-mortem that other muscles besides the abductors, namely, the arytaenoideus and the crico-arytaenoidei laterales, both of which adduct the vocal cords, were very much wasted, though apparently not so much as the abductors. Unfortunately accurate observation of his larynx was not made lately; he was very difficult to manage with the laryngoscope, and I confess that I was unable to get a good view of his glottis in the last weeks of his illness; but early in his case the features were characteristic of paralysis of the abductors only, the cords being together in the median line with only a narrow chink of about one twentieth of an inch between them; phonation was almost perfect, and up till February 13th it was stated he could speak clearly. It seems probable that it was after this that the adductors became most affected when the alteration in the condition due to their paralysis for various reasons was not closely watched. It is interesting to note that of the twenty-two cases collected by Dr. Semon, in three there was atrophy of other muscles, though the abductor paralysis was the prominent clinical feature. In one the adductors were degenerated, in another the left thyro-arytaenoid, and in a third the left crico-thyroid muscle; so that we cannot claim for the case that it is one in which the abductors were alone diseased, but one in which they were earliest affected, as shown clearly by the symptoms with which he first presented himself. In a number of cases of abductor paralysis collected by Burow the condition of the voice was by no means uniformly perfect, though the group of symptoms characteristic of this form of paralysis includes free expiration

and a clear voice. Of twenty-five cases in which the state of the voice is recorded, it was normal in twelve, hoarse in eleven, and absent in two.

The following is a case of the same nature, but differs from the preceding in that the affection was not the same on the two sides, and that the clinical features were complicated by the symptoms due to tracheal stenosis. It is, however, a good illustration of the early implication of the abductor fibres as a result of a lesion of the vagus or recurrent nerves. The laryngoscopic examinations were neither long, nor frequent, nor easy, but it was clear that the left cord was paralysed, and the right did not move perfectly freely. At the post-mortem examination both recurrents and vagi were found involved in the growth, but the left recurrent was smaller than the right, and the left vagus was much pressed upon. With all this it was the left posterior crico-arytænoid muscle alone that was atrophied.

CASE 2.—Lympho-sarcoma of anterior mediastinum involving both vagi; obstruction to left innominate vein; atrophy of left posterior crico-arytænoid muscle; stenosis of trachea. Death.

(Reported by Mr. T. B. WINTER.)

Hugo A—, æt. 49, was admitted under my care on March 18th, 1882. He was a Finlander by birth, had been a master of a ship for ten years, and was now clerk in a shipbroker's office. His parents died above seventy years of age; he has a brother and sister living and well. He is not married. Twelve years ago he contracted syphilis, having a chancre and sorethroat, and he has now, at the upper border of the sternum, a scar an inch and a half to two inches in diameter, which was produced by a blister applied at this time. Four years ago he had congestion of the left lung. He has had gonorrhœa three or four times; never rheumatism or gout. About twelve years ago he gradually lost the sight of his right eye. Six weeks ago he caught a severe cold, gradually became hoarse, had cough and great pain in the chest, and spat up a good deal of phlegm. The hoarseness gradually improved, so that after

keeping his room for ten or twelve days he was able to resume his work, although the pain in the chest, neck, and shoulders got worse. After two or three days he again had to take to his room, and about ten days ago he noticed that his throat was beginning to swell. He has lately become much weaker. He was under the care of Dr. J. De L. Temple, who obtained his admission into the hospital.

Present condition.—He is a very fine, strongly-built man, with broad, deep chest, and moderate amount of fat. He complains of pain in the chest and difficulty of breathing. The pain is situated over the sternum, in the chest, and below the shoulder-blades, especially on the right side. The dyspnœa is associated with a moderate amount of stridor; he breathes twenty-five times in the minute in a gasping manner. From time to time aggravations of the difficulty occur, in which he takes a succession of deep gasping respirations, followed by short expirations; the head is thrown back, the eyes become prominent, and the face, at all times rather livid, becomes darker still. On examination of the throat the fauces and back of the palate are very congested, of deep red colour, much swollen, and sensitive to touch. The laryngoscopic examination is on this account somewhat difficult, but shows that the epiglottis, ventricular bands, and surrounding mucous membrane, now brought into view, are similarly congested, and that the vocal cords themselves are slightly reddened. The left cord is immovably fixed in the middle line, the right moves with respiration and phonation, but does not diverge to its full extent.

In the right posterior triangle of the neck there is a large lobulated growth, passing behind the clavicle downwards into the chest, and there is a similar smaller growth on the left side. There is markedly higher pitched note on percussion below the inner ends of the clavicles, especially the right; the line of dulness slopes from the lower part of the sternum up to the middle of the clavicle, and similarly on the left side to the inner end of the clavicle. Over this area there is high-pitched, almost whistling, tubular breathing; over the same area there is distinct transmission of whispered voice sounds, but no increase of vocal resonance; this is most remarkable, and is exactly limited by the line bounding the dulness. In this

position the tracheal resonance is abnormally distinct and of a whistling character. There is no pulsation over the dull area or over the sternum. In both suprascapular fossæ behind there is dulness and whistling tubular breathing. Air enters both lower lobes fairly well though not perfectly. The chest does not expand freely, but there is no evident suction action. The venous radicles over the sternum are enlarged and varicose, and the condition is limited to the radicles of the internal mammary vein. There is no obstruction to the cervical veins. The heart's impulse and sounds are normal. The urine is high coloured, acid, sp. gr. 1032, with an abundant red deposit of lithates, and no albumen.

On the day of his admission Mr. Jacobson saw him with me in reference to the possibility of tracheotomy being at some future time required, though it was known that tracheal pressure was extremely probable. He was ordered 20 grains of iodide of potassium three times a day, 8 grains of calomel at once, to be followed by 2 grains with a quarter of a grain of opium every six hours. He had a diet of milk, beef tea, two eggs, and farinaceous food, and was placed in a steam-tent.

During the next day or two he was very sick.

March 23rd.—He is salivated, the gums are sore, the breath fetid, and the tongue slightly swollen. With the laryngoscope the glottis is still to be seen very narrow; the epiglottis is not quite so red; the uvula, soft palate, and mucous membrane of the hard palate are swollen, red, puffy, and soft to the touch. The medicine is omitted. On examining the chest, it appeared to me the dulness was less than represented in the former note, and that the pectoriloquy was much less sharply limited, gradually rather than suddenly diminishing. I examined carefully with a view to localising the stenosis which caused the stridulous breathing, so as to ascertain what share the trachea, and what the glottis, had in it; but though the sounds diminished as one got away from the trachea to the chest, it was impossible to observe any appreciable difference on going upwards from the trachea to the larynx. There is slight suction action at the back of the lower ribs on the left side. Resp. 22, pulse 100, temp. 99°.

25th.—The swelling on the left side of the neck has considerably increased the last few days.

28th.—The voice is now reduced to a hoarse whisper. He expectorates a quantity of offensive frothy mucus. There is some ulceration of the mouth behind the molar teeth of the lower jaw. Ordered 10 grains of chlorate of potash three times a day and a chlorate of potash gargle.

30th.—Abundant expectoration of thick yellow sputa, mixed with much frothy saliva; the odour is not so offensive as before. Morn. temp. $97\cdot4^{\circ}$, even. temp. $97\cdot5^{\circ}$.

31st.—The respirations were to day 36, pulse 94.

April 2nd.—About 10 a.m. he became much worse; he had severe dyspnœa and profuse perspirations. He was slightly relieved by $1\frac{1}{2}$ ounces of brandy by the mouth, and a subcutaneous injection of 15 minims of ether. But at 1 p.m. he was again worse; the temperature fell to 95° , he became comatose, and died quietly between 2 and 3 p.m.

Tracheotomy was not attempted, and, as the event showed, would have been useless. The post-mortem was made by Dr. Goodhart.

All the glands occupying the parts above the clavicle were large and fleshy. On section they gave a copious creamy juice. They were continuous with a large mass of a similar character which surrounded the trachea in the mediastinum. This extended downwards to just above the valves of the heart, more on the right side than the left. It practically completely surrounded the trachea from the second ring, to one and a half or two inches above the bifurcation, and in the mediastinum it had all but occluded the upper part of the superior vena cava by pressure, and the left innominate vein was completely occluded by a greenish, decolorised clot. The œsophagus was also distinctly pressed upon, being distended over the tumour which lay in front of it, and its anterior wall was indeed adherent to the growth. The pneumogastrics and recurrent laryngeal nerves ran into the mass on both sides, or had masses of glands firmly adherent to them. And on the left side the main trunk of the pneumogastric nerve was spread out over the growth. Both recurrent laryngeal nerves appeared to be very small, but the left decidedly the smaller.

The larynx appeared to be quite healthy except that there was *considerable wasting of the left posterior crico-arytenoid muscle*. In slitting open the trachea from above, and making transverse

sections of it below, the following state of things showed itself. The mucous membrane from the second ring downwards was much thickened and white from infiltration of the tissue by new growth, and in addition, at the upper part on the left side, was a lobulated gelatinous mass protruding inwards into the canal and considerably reducing the calibre of the tube.

Lower down there was simple diffused infiltration, which had reduced the tube to a quarter of an inch transversely, and three eighths of an inch antero-posteriorly. This condition extended to about an inch and a half above the bifurcation. The tracheal rings were all extensively ossified, and the anterior section of all the tissues in front of it showed that the thyroid gland was in part replaced by the same white growth as existed in the mediastinum. Indeed, Dr. Goodhart thought that possibly the growth might have begun in the thyroid and spread outwards and downwards. The lobulated mass fungating into the trachea was certainly very like a thyroid growth, and with this opinion Dr. Fagge coincided, saying that the tracheal affection was very like a specimen that had been put up in the museum the preceding year.

The liver was studded with white, soft, creamy masses of all sizes, and weighed 110 ounces. Some of the portal glands were considerably enlarged, surrounding the portal vein and bile-ducts, but not pressing upon them.

The kidneys weighed 12 ounces, contained numerous large cysts, but were otherwise healthy, and had smooth surfaces.

The peritoneum, stomach, intestines, gall-bladder, pancreas, suprarenal capsules, and spleen were healthy.

The next case, imperfect though it is, is very interesting, from the fact that the paralysis of the abductors, from which the patient at one time nearly lost his life, almost completely recovered. There can be little doubt that syphilis was the remote cause of his disease; this conclusion resting, not alone on the history of chancre and the presence of scars and ulcers on his lower extremities, but on the peculiar course and progress of the special nerve symptoms.

The tendency of syphilis to produce lesions scattered over the nervous system, so that the most independent and different

parts, whether centre or nerve trunk, may be at some time or other affected, is well known. And in this case at least three lesions were produced more or less independent of another, viz. the laryngeal paralysis which recovered, the dysæsthesia of the third division of the left inguinal nerve, and the hemiplegia, with which also the pains in the arm may be associated.

Again, making use of Dr. Semon's list of cases we find that about half of them are due to peripheral pressure of the vagi or recurrents by different growths, namely, five cases of aneurism, three of enlargement of the thyroid, two of connective tissue in connection with chronic lung disease, one of enlarged bronchial glands, and one of cancer of the œsophagus. The others are, with one exception, cases in which the laryngeal paralysis has been ascribed to a central, or at least an encephalic cause, and has often been associated with other symptoms of a general paralytic nature, or such as indicate disseminated sclerosis or syphilitic lesions. The fact of this patient improving so considerably as he did, may also be held as confirming somewhat the evidence of its having a syphilitic origin; not, I will allow, very strongly, because of these nine central cases there are seven whose histories are not complete, *i.e.* either they were still living at the time of the report, or if dead, no post-mortem examination had been made; and until we know everything about these lesions it would not perhaps be right to say they are incurable. But it is remarkable that only one of the nine cases improved, and that was one that was diagnosed as syphilitic and was treated with iodide of potassium.

Another possible explanation of his recovery would be, of course, that the disturbance was of a functional or hysterical nature. It has been already said that abductor paralysis is nearly always organic, but cases have been recorded of an obviously functional nature, with all the characters and associations of hysterical complaints; for instance, the female sex, previous hysterical attacks, and sudden development, or sudden cure or relapse of the laryngeal symptoms. But these characters and associations are wanting in the case under consideration, and the ultimate death of the patient in connection with his paralytic symptoms seems to exclude such a view.

CASE 3.—*Paralysis of the posterior crico-arytænoid muscles; tracheotomy; almost complete recovery; left hemiplegia. Death.*

(Reported by Mr. MILLIGAN and Mr. G. N. PITT.)

John S—, æt. 48, was sent up to the hospital in September, 1880, by Dr. Steele, of Reigate, on account of stridulous breathing, and I was asked to see him by Mr. Howell, the house-physician. On examining him with the laryngoscope; it was at once obvious that the abductors of the vocal cords were paralysed. He was not prepared to come into the hospital, and remained under the treatment of his own medical attendant, being subsequently for three weeks in the Cottage Hospital at Redhill. However, he got so much worse that he sought admission at Guy's, where he was received on October 25th, under the care of Dr. Wilks, to whom I am indebted for permission to use this case. The following history was taken by Mr. R. A. Milligan. The patient has been a soldier, but since leaving the army has been a painter. He has a good family history; he is married and has nine healthy children, and his wife never miscarried. With the exception of a chancre, which he had many years ago, he has been well till six months ago when his present illness began with hoarseness of voice and a peculiar noise whilst breathing. For three months the noise has been so great that the neighbours on the opposite side of the road could hear it. The dyspnœa has been variable; at times he seemed pretty well and could breathe easily, but at others he could hardly get his breath, except with the greatest difficulty. He has been treated by steam inhalations and the use of the galvanic battery. He has had no difficulty in swallowing except what is due to the interference with respiration.

On admission he was suffering from dyspnœa with stridor, and so bad was he that no examination could be made of the chest. At midnight the obstruction to the entry of air had increased, so that tracheotomy had to be performed, and it was done under chloroform by the house-surgeon. Considerable hæmorrhage occurred both at the operation and two or three hours after. It was restrained by the use of the tracheal dilator devised by Mr. Golding-Bird, with plugging by lint.

On the following day his breathing was easy, and the dilator was replaced by a vulcanite tube; and this two days later by one made of rubber.

On November 3rd there was a little bleeding, and examination of the chest showed a little tubular breathing and crepitation at the right base, but these quickly cleared up, and on November 13th he was removed from the steam-tent to a bed in the open ward. Towards the end of the month he began to talk and to breathe through the larynx, and on December 7th it is stated he can go about with the tube closed for the greater part of the day, but he left it open at night. He left on January 4th, wearing a red rubber tube. Dr. Mahomed examined him on this day, and found tubular breathing and bronchophony at both apices; he thought they might have been the result of transmission of tracheal sounds by indurated and thickened connective tissue at the root of the neck. There was no other evidence of any disease in the chest. The radial pulses were soft, natural, and equal in all respects.

I saw this patient again on June 5th, and made the following note:

He still wears the red rubber tube, but he has cut it short, about the middle of its curve. He removes it every day to clean it, and if he leaves it out long, has some difficulty in replacing it.

For a month after leaving the hospital he continued to close the tube during the day and to leave it open during the night; but for more than a month he has had it completely stopped both day and night. He breathes easily, even during considerable muscular exertion, and he talks easily and loudly, though not with perfect clearness. On examination with the laryngoscope, the vocal cords are seen to diverge considerably with deep inspiration though not quite to their full normal extent; on phonation they are almost perfectly approximated.

He continues his occupation as a painter, has a fair appetite, and swallows well. For two months, however, he has had numbness, and a burning sensation over a portion of the lower lip and adjacent chin, running from the middle line for two inches to the left. He has also had since leaving some pains in his arm, that he calls rheumatic.

On July 23rd he was again admitted, this time under Dr Moxon's care, into Guy's Hospital. Some weeks before he had numbness in the left hand, and frequently dropped things; and a day or two later he was seized with unconsciousness. This lasted about ten minutes, he then crawled up to bed, where he slept for an hour or two, and on waking found himself hemiplegic on the left side. He stated also that he had dimness of vision with his left eye as well as diplopia; the latter had disappeared, the former was improved. On admission he had a moderate degree of weakness in the left arm, leg, and face. The voice is said in the report to have been lost, and the arytaenoid cartilages moved freely; he could cough efficiently; the palate, uvula, and pharynx were found to be somewhat anæsthetic. It is on this occasion noted that he had several pigmented circular scars half an inch in diameter on the extensor surfaces of both legs, and two recent sores on the left leg above the inner malleolus. Measurements of the limbs showed that the left arm and leg were each half an inch less in circumference than their fellows. The urine was free from albumen, and the optic discs were normal.

He remained in the hospital a month, taking twenty grains of iodide of potassium three times a day, and during the last ten days $\frac{1}{32}$ gr. of perchloride of mercury in addition. The upper extremity wasted considerably, and he went out unrelieved.

About a month later he died rather suddenly at his own home, and no post-mortem examination was obtained.

The following case presented itself among my out-patients, and was admitted into the ward.

No cause could be assigned for the symptoms except that she had caught cold, but there was no evidence of catarrh on laryngoscopic examination; and whether it had actually existed, or the patient only mistook for it the paralytic phenomena, it is impossible to say. As in other cases her stridor was much louder at night. After being in the ward a few days she left at her own desire, and subsequent efforts I made to her trace her to home failed completely; and the case is unfortunately very incomplete.

CASE 4.—*Paralysis of the abductors of the vocal cords.*

Elizabeth B—, æt. 32, was admitted under my care March 28th, 1881. She is married, and has had no children, but two miscarriages. Her family history is good and she has always been well herself. Two months ago she caught a cold, having a feeling of tightness in the chest with a dry hacking cough. After these symptoms had lasted about a week she almost completely lost her voice, and had difficulty in breathing. The dyspnœa has increased since then, but the voice has somewhat improved. She has lost flesh a little.

On admission, she is a healthy, strongly built woman, with warm, dry skin, and no cyanosis. She has a good appetite without vomiting or nausea, and no dysphagia, nor is any coughing excited by swallowing. She has dyspnœa both in inspiration and expiration, the stridor being louder with inspiration, and so noisy at night as to disturb all the other patients; sometimes she starts up at night with a sensation of being choked. The dyspnœa is increased by exposure to cold air. She speaks in a harsh whisper, and has a dry, hacking cough, with a very harsh, croupy noise. Laryngoscopic examination shows that the cords are close to and parallel to one another, perfectly white and healthy themselves. There is only a narrow slit between them, and there is no alteration in their position on deep inspiration.

Respiration is thoracic, and there is no sucking in of the supraclavicular spaces or false ribs. The dyspnœa is not accompanied by any pain, but she has a tight feeling in the chest, especially at the top of the sternum, as if the obstruction lay there.

The chest is well formed, resonant on percussion; air enters freely everywhere, but laryngeal stridor is heard all over the chest. There are no signs of aneurism or intrathoracic tumour. The impulse of the heart is not well defined; the sounds are normal; præcordial dulness rather diminished. Pulse regular, full, compressible. Urine normal.

She only stayed in five days, leaving on account of family matters at home. The dyspnœa at night was considerable, and was unrelieved by steam inhalation.

It has been above mentioned that a contrast may be drawn

between abductor and adductor paralysis in regard to their origin, and that the former are nearly always organic. Dr. Semon¹ says: "Although there are a few cases of hysterical paralysis of the *abductors* on record (Fraenkel, Guttmann, Biermer, Burow, Schreiber, Mackenzie-Semon), yet the immense majority of cases of 'hysterical paralysis' hitherto recorded concern the *adductors* (and tensors) only!"

The following case appeared to me at the time I saw it to be clearly one of functional (or hysterical) paralysis of the abductors, as it occurred in an overworked, but otherwise perfectly healthy, young woman; it came suddenly and lasted but a few hours, recovering spontaneously, for she had but a dose or two of the medicine. On the other hand, the duration, which was too short for organic disease, was too long for one to entertain the idea that it was simply spasm of the adductor instead of paralysis of the abductors. Spasm occurs suddenly and subsides quickly and completely, the paroxysm lasting a few minutes at the utmost.

CASE 5.—Dyspnœa and inspiratory stridor. Rapid recovery.

On May 5th, 1879, I was asked to see one of the nurses in our clinical ward for men. She was a most attentive and energetic nurse, but had been considerably overworked in consequence of the number of severe cases in the ward. While out of the hospital this evening after her duty was over, she was taken suddenly ill with soreness of the throat and difficulty of breathing. I saw her at midnight; she said that she was better than she had been, but she appeared exhausted and alarmed, and was breathing with some stridor. The stridor was only during inspiration, expiration was clear, and cough and the voice were natural. The tongue was clear, the back of the pharynx rather pale, and there was neither inflammatory redness nor membrane. With the mirror the epiglottis was seen to be natural, and the vocal cords bright white, but they were lying close together as though from paralysis of the abductors. She was ordered some medicine containing strychnia and iron, and the following morning, after a night's rest, was perfectly well.

Is it not possible that cases such as the above would be

¹ Loc. cit., p. 22.

found to be much more frequent if the laryngoscope were more freely used? I have, at least, seen one similar case since. But those to which Dr. Semon in the before-mentioned passage alluded, were of much longer duration, and therefore much less easily differentiated from those due to an organic cause. Dr. Semon was kind enough to furnish me with the complete references to these cases, and it seems to me that there is nothing in the intrinsic features of the paralysis itself to distinguish it from that which is demonstrably due to an organic cause. The dyspnœa, the inspiratory stridor, the free expiration, the condition of the voice, either perfect or somewhat hoarse, are the same, and the laryngoscope shows the same close approximation of the cords, which are drawn even closer during inspiration, but do not separate widely in expiration. The diagnosis appears to rest either upon the coexistence or previous occurrence of obviously hysterical symptoms, upon the absence of symptoms pointing to an organic lesion, upon the variability in the degree of paralysis, or upon its sudden or rapid recovery.

To these cases the following much more closely approximates, only that the duration of the paralysis was still very much shorter. The patient, a girl, was highly hysterical, and the laryngeal paralysis lasted about six days.

CASE 6.—*Hysteria ; catalepsy ; dyspnœa and inspiratory stridor. Recovery.*

(Reported by Mr. NEWNHAM, M.B., and Mr. RYLE.)

Flora P—, æt. 12, was admitted under my care in July, 1881, for hysterical catalepsy. This first appeared in March, in the midst of perfect health, and with no apparent cause except a fright six weeks previously. She became suddenly rigid, with fixed eyes, as though unconscious, and then commenced screaming, "Oh my head, my head." This continued two hours, when she slowly recovered. During the night she had several fits of the same description without the screaming, and such attacks have been repeated, often several times in the day, until the present time. In each fit she used to become rigid, the eyes were fixed, and she was insensible to pricking and pinching. She often muttered, sometimes carried on a rational conversation, and has been known to sing, write, read, do

arithmetic on the slate, sew and crochet during the continuance of the attack. She was a pale, emotional child; she had several fits of the kind described. The limbs were rigid, but easily took up any new position, and remained in it for a long time. After a few days the fits diminished in frequency, and she went out on August 10th.

On October 5th she came amongst Dr. Goodhart's out-patients, suffering from inspiratory stridor. The larynx was examined, and the vocal cords were seen to be in close apposition. The stridor had been noticed about three days. Dr. Goodhart asked me to see her, and she was admitted under my care. Besides the paralysis of the cords, the parts were found to be remarkably anæsthetic, and the tonsils were enlarged. After a few days the stridor ceased entirely. She had one or two fits of short duration, and was sufficiently well to leave on October 19th.

While both of these cases differ strikingly from the few published cases of functional abductor paralysis in the short duration of the affection, though this alone can scarcely remove the affection from the category of paralysis, another doubt has presented itself to me on finding that symptoms somewhat similar may arise from what is described as a "perverse action of the vocal cords" without actual paralysis, functional or otherwise, of the abductors. In this condition¹ there is inspiratory stridor and dyspnœa from approximation of the processus vocales and vocal cords, but during expiration, or at any rate for a short period during each act of breathing, the vocal cords are separated by the action of the abductors. I regret that the short notes I have of these two cases do not give more details of the behaviour of the vocal cords, so that it is impossible for me to assert from them alone that in these cases the condition, so long as it lasted, was one of abductor paralysis, and not merely the "perverse action" newly brought to our notice. While therefore, allowing that what is not looked for may be readily overlooked, my strong impression is that the cords were fixed near the middle line during both inspiration and expiration. Imperfect as the cases are, I venture to think they are of interest as

¹ Semon, German translation of Morell Mackenzie's 'Diseases of the Nose and Throat,' vol. i. Fraenkel, 'Berl. klin. Woch.' 1878.

illustrating the fact that dyspnœa and inspiratory stridor, from approximation of the vocal cords, may occur as a temporary affection, which is not spasmodic, and has not for its remote cause an organic lesion, but probably some condition of nerve failure, as hysteria or exhaustion.

CASES
OF
MULTIPLE SMALL ABSCESSSES OF THE
LIVER.

BY R. E. CARRINGTON, M.D.

I HAVE ventured to group together the succeeding series of cases, both because they seem to be of extreme interest in themselves, and also because such cases generally, I believe I may say invariably, present very considerable difficulty in diagnosis; further, although not of extreme rarity, they are certainly not common. With regard to the point of frequency, I have not the numbers of all the cases which are noted in the post-mortem records of Guy's, but it will be some indication if I mention that there were two instances in 1874, two in 1875, two in 1877, and two in 1878. These are not, of course, all the cases of suppuration of the liver which have occurred in these years, but only those, the nature of which I shall state hereafter, and with which it is the purpose of my paper to deal.

I am afraid the histories will appear to have been given with unnecessary prolixity of detail, but I have judged it best that they should be complete, in order that their obscurity from a clinical point of view may be apparent.

It will, I think, be generally admitted that to differentiate the cause of an irregular pyrexia is often a matter of much perplexity. Of course the more perfect our knowledge of all possible causes of such a condition, the greater will be our chance of arriving at a correct conclusion; and I venture to

suggest that cases of the condition under review are of sufficient importance and frequency to be borne in mind in endeavouring to clear up the nature of a particular case. It is not, of course, intended to suggest that the affection is one not already perfectly well known, nevertheless as I have been fortunate enough to have seen most of the examples of which I have detailed the histories, and have thus had prominently presented to me the extreme difficulty they have frequently caused in diagnosis, I have ventured to think that by grouping them together, and comparing their points of similarity and of difference, some facts of clinical interest might be brought out.

The condition underlying all of them was the presence throughout the whole or part of the liver, of many, often innumerable, small points of suppuration, and though it may be with good reason objected that the pathology of all was by no means identical—that the pus in some cases was situated in the portal vein, in others in the bile-duct, and in others indifferently in the hepatic substance—still they all agree in the presence of multiple small abscesses in the organ, and to these the symptoms were due, apart from such as were caused by a primary lesion, when present.

All examples of this condition will probably fall under one of three heads, viz.:

Cases in which no primary cause can be ascertained.

Those in which there is some source of infection in the distribution of the radicles of the portal vein.

Those in which there is some persistent obstruction to the outflow of bile.

The first two groups may possibly be associated together. It is difficult, if not impossible to fix the date of the onset of suppuration, but I have done this as closely as possible, and I have given the duration of each case hereafter. From this it will appear that although no doubt the most acute cases occurred in the first group, yet that no distinction can be made on the score of duration between this and the second, for all the cases presented the greatest possible variation in this respect, the limits being five and eighty days.

The group in which the cause of the suppuration is not ascertainable will probably always constitute a small proportion of the whole; nevertheless, Cases 1, 2, and 3 are examples

of this class, and therefore it cannot be disregarded; still, undoubtedly it will generally be a fact that we shall often have the history of some antecedent illness to guide us in diagnosis. So far as the instances detailed go, it would appear that the affection is invariably fatal; Case 10 is, however, very important, as suggesting that the morbid condition may go on to cure. I have notes of another case pointing in the same direction, but I shall revert to this question again hereafter.

I propose now to briefly analyse the appended reports, so as to ascertain the most constant symptoms which may guide a diagnosis, and I shall first take the actual condition presented by the patient, and then enter into the subject of causation with a view to see how far this has been revealed by symptoms antecedent to the suppuration.

I.—Duration of illness.

Case 1. 60 days.

Case 2. 64 days.

Case 3. 5 days and 2 or 3 hours.

Case 4. 47 days.

Case 5. 24 days 18 hours.

Case 6. 75 days.

Case 7.

Case 8. 18 days 20 hours.

Case 9. 48 days.

Case 10.

All the preceding are examples of the infective origin; the succeeding were obstructive.

Case 11. 37 days.

Case 12. 80 days.

Case 13. Probably suppuration existed the last few weeks only, the illness lasting six months.

It will thus be seen that there was the greatest possible variation in point of duration.

II.—Aspect of patient.

Case 1. Was exceedingly low, and did not speak above a whisper, wasted, with eyes sunken, breathing hurriedly.

Case 2. Very ill, was thin, pale, and looked ill.

Case 3.—Looked exceeding ill. Walked into the ward supported by one man. Eyes sunken. Facies Hippocratica. Gave his answers excitedly, but always the same dates. Very restless. Knees sometimes flexed, sometimes extended.

Case 4. Emaciated. Eyes bright.

Case 5. Lay on her back and moved with difficulty. Constantly moaning. In a sleepy, dreamy condition, answering with difficulty and in monosyllables. Face flushed, with pained expression. Semicomatose five days later. Just before death, cardiac pain and restlessness.

Cases 6 and 7. No mention.

Case 8. Before admission looked feverish, and face was flushed. Lay in bed on right side with knees drawn up, looking ill and in pain. Eight days after delirium, restlessness, subsultus.

Case 9. Like a case of fever. Very apathetic and drowsy. Lay on his back taking little notice. Could be roused to answer questions, but rapidly sank into apathy. On thirty-fourth day of illness, wandering and restless.

Case 10. Thin, pale, and anæmic.

Case 11. No mention.

Case 12. Cachectic. Lay low down in bed "all of a heap," knees drawn up. Constantly uttering low short groans, and now and then placing hand on abdomen as though in pain. Eyes closed and extreme apathy. Nocturnal delirium.

Case 13. Always cheerful. Daily expressed herself as feeling better. Never delirious. On day of death lying in a calm, unconscious state, like sleep.

The prominent characteristics appear to be, that the patient looked extremely ill; that the illness appeared to be abdominal, and in connection with this it is to be noted that local or general peritonitis was commonly present. A deep apathy was frequently present.

III.—*Wasting.*

Case 1. Wasted.

Case 2. Thin.

Case 3. "Says he has lost two stones during the week." Autopsy.—"A muscular man."

Case 4. Somewhat emaciated.

Case 5. Much wasted.

Case 6. Body spare.

Cases 7 and 8. No mention.

Case 9. Body fairly well nourished.

Case 10. Wasted. His extensive abscesses, other than hepatic, would account for this.

Case 11. Body fairly nourished.

Case 12. Progressive wasting, from five weeks prior to admission. Autopsy.—Body emaciated.

Case 13. Steady and progressive emaciation.

Wasting was thus prominent throughout.

IV.—*Sweating.*

Cases 1 and 2. No mention.

Case 3. None before admission. On sixth day profuse sweating from 9 p.m. to midnight.

Cases 4 and 5. Hot and dry skin on admission.

Cases 6 and 7. No mention.

Case 8. Before admission "hot and feverish." On seventh day skin hot and perspiring.

Case 9. No mention.

Case 10. Extensive suppuration, besides that in the liver.

Case 11. No mention until the ninth day in hospital, when she woke up sweating.

Case 12. On admission, skin warm and in parts moist. No further mention.

Case 13. Last few weeks "copious offensive perspiration."

It would appear that sweating, though present at times, was neither a constant nor prominent symptom.

V.—*Appearance of abdomen.*

Case 1. Distended with fluid.

Case 2. Tumid.

Case 3. No general distension. Respiration more thoracic than abdominal.

Case 4. Abdominal muscles very tense. Forty-fifth day, great distension.

Case 5. Respiration thoracic.

Case 6. Much ascites.

Case 7.

Case 8. Hard with rigid recti. Fifth day, abdomen less tumid; he was under the influence of opium.

Case 9. Natural. Thirtieth day, swelling on right side below ribs. Thirty-fourth day, very obvious increase of swelling.

Case 10. Obvious abscesses in left loin and right groin.

Case 11. Abdominal muscles particularly tense.

Case 12. Somewhat distended. Forty-first day, neither hard nor tender. Forty-seventh day, there appeared to be a slight bulging of side of chest in hepatic region, but measurement only showed a difference of half an inch. Fifty-fourth day, full but no fluctuation.

Case 13. Nothing noticeable.

Abdominal distension was thus present in eight out of eleven cases (Cases 7 and 10 being omitted). It occurred with general peritonitis or ascites in four, with local peritonitis about the liver in three, and with old adhesions about the liver in one. Of the three cases in which it was absent in two there was local hepatic peritonitis, and in the same two thoracic respiration was noted. This last was a symptom which the late Dr. Murchison laid stress upon in the diagnosis of peritonitis.

VI.—*Enlargement of the Liver.*

Case 1. Weighed $67\frac{1}{2}$ oz., so that it was slightly enlarged; no mention is made of it in the clinical report.

Case 2. No mention.

Case 3. Dulness from fifth rib to a finger's breadth below the margin; weight 98 oz.

Case 4. Dulness to level of umbilicus.

Case 5. Dulness much increased.

Case 6. Weighed 67 oz.; organ was contracted by fibroid material.

Case 7.

Case 8. On admission, dulness did not extend below the ribs; on seventh day after it was $1\frac{1}{2}$ in. below.

Case 9. On admission, dulness reached from between fifth

and sixth ribs to two fingers' breadths below the margin. It increased so as to reach the level of the umbilicus.

Case 10.

Case 11. On admission, dulness from fifth space to one inch below the margin; eleven days after, liver of stony hardness 2 in. below ribs.

Case 12. Dulness from third rib to one finger's breadth below margin; eleven days after there was increase in size of hepatic region of half an inch; eighteen days after the dulness had increased an additional finger's breadth downwards.

Case 13. None.

Thus the liver was enlarged more or less in every case but the last.

VII.—*Abdominal pain and tenderness.*

Case 1. Illness commenced with both. On admission abdomen was tender.

Case 2. Pain for about one month before admission.

Case 3. Pain up to admission. There was no general tenderness, but pain was produced over the liver by pressure beneath the ribs, and also over the spleen; the day after there was slight pain and tenderness. This seems the most fit place to call attention to the peritoneal rub, which was discovered by Mr. C. J. Symonds (then Clinical Assistant), as it afforded direct evidence of inflammation on the surface of the liver.

Case 4. Pain throughout. Three days after admission abdomen tender all over; twenty days after this great abdominal distension and tenderness.

Case 5. Moaning and complaining of pain. Hepatic region tender.

Case 6.

Case 7.

Case 8. Both. Percussion of liver caused intense pain.

Case 9. On admission, tenderness over liver, which persisted throughout.

Case 11. On admission, throbbing pain across front of lower part of chest, continuous for hours. On fourth night much pain. On twelfth day great pain in right side, hepatic region very tender.

N.B.—The liver was probably tender on admission, for a note prior to the preceding states it to be *still* so.

Case 12. On admission, abdomen tender, posture as of one in abdominal pain, often placing her hand on her abdomen. No hepatic tenderness. Six days after, neither hardness nor tenderness. On 11th, more tenderness in right hypochondriac region than elsewhere, and subsequent notes all indicate abdominal pain and hepatic tenderness.

Case 13. Neither.

Abdominal pain and tenderness, especially in the hepatic region, appeared in every case except the last. It was associated in all but this case with general or hepatic peritonitis, old or recent.

VIII.—*Condition of tongue.*

Cases 1 and 2. No mention

Case 3. Dry, rough, and horny on sixth day, and continued so to the end.

Case 4. Creamy fur on admission, twenty-one days later a slight fur is noted.

Case 5. Dry brown on dorsum, pale at edges. Sordes on teeth and gums.

Cases 6 and 7.

Case 8. On admission, covered with white fur, lips dry; fourth day, creamy white fur, and again on sixth.

Case 9. Dry, furred in the centre with red edges. This appears to have been constant throughout.

Case 10.

Case 11. No note.

Case 12. On admission, lips and tongue dry, sordes on teeth; and this, with modification in degree, was the case throughout.

Case 13. No note.

Thus it appears that a dry, brown, or furred tongue with sordes on the teeth, was pretty constant.

IX.—*Vomiting, diarrhœa, or constipation.*

Case 1. Vomiting and constipation throughout.

Case 2. Vomiting and diarrhœa prominent.

Case 3. No sickness. Constipation.

Case 4. Diarrhœa on two occasions. Vomited blood one week before admission, vomiting fourteen days after, and six days after this of blood-stained fluid twice in the day.

Case 5. Vomiting and diarrhœa prominent throughout.

Case 6. Diarrhœa.

Case 7.

Case 8. Vomiting before, but not after admission, and constipation before; but a daily action of bowels, the motions not relaxed after coming in. Opium had been given.

Case 9. No vomiting, but diarrhœa throughout (dysentery).

Case 10.

Case 11. Vomiting prominent throughout. Ejecta revealed bile to tests and were very bitter. Constipation.

Case 12. No vomiting. Constipation.

Case 13. No vomiting. Bowels regular.

Vomiting was present in half the cases; and of eleven cases, in one the fæces were natural except in being clay-coloured, in five there was constipation, and in an equal number there was diarrhœa. These conditions were connected with obvious causes, such as dysentery, gastric ulcer, and peritonitis in most cases.

X.—Rigors.

Case 1. Repeated throughout illness.

Case 2. No mention, probably absent.

Case 3. None.

Case 4. Illness appeared to commence with "cold shivers" lasting for six hours, no subsequent mention.

Case 5. Cold and shivering the day before death.

Case 6. Occasional after the ulceration, which was probably the cause of the hepatic suppuration, and also before.

Case 7.

Case 8. None.

Case 9. None.

Case 10.

Case 11. Hot and cold flushes and severe rigors during last seven days of illness.

Case 12. None.

Case 13. None.

Rigors were far more often absent than present.

XI. *Jaundice.*

Case 1. Face yellowish and sallow, but conjunctivæ pale. The only evacuation was clay-coloured.

Case 2. None.

Case 3. None.

Case 4. Complexion sallow. No bile in urine.

Case 5. None.

Case 6. Two months before admission an attack of biliary colic with jaundice. No mention is made of jaundice on admission, nor at the autopsy; the liver was fawn-coloured.

Case 7.

Case 8. Intense, of skin and conjunctivæ. Urine contained bile. Fæces "dirty white."

Case 9. None.

Case 10. Yellowish, pallid complexion. Conjunctivæ pale.

Case 11. On admission conjunctivæ jaundiced. Urine examined four times and always contained bile. At autopsy no jaundice.

Case 12. Jaundice and clay-coloured motions five weeks before admission. When seen skin had a slight jaundiced tinge, urine showed bile, and fæces were clay-coloured, and the yellow tinge of the skin is mentioned throughout.

Case 13. Intense.

Of twelve cases jaundice was intense in two, one without obstruction of the ducts, and one with complete obstruction. It was slight in two others, and in each of these the urine contained bile; there was obstruction in both of these. Perhaps Case 1 should be grouped with these in spite of the pale conjunctivæ. The complexion was sallow or yellowish in two cases without other evidence; and in all the others jaundice was absent. This symptom was therefore only present in a third of the cases.

XII.—*Temperature, pulse, and respiration.*

Cases 1 and 2. No thermometrical record of these cases is preserved, but in Case 1 it was noted that the skin was cool.

It is well known, however, how deceptive a mere appeal to touch may be.

	Temp.	Pulse.	Resp.
Case 3. Febrile.	Highest 106·2°	192	44
	Lowest 102·8°	132	28

Case 4. Febrile, but once

98·4° ; otherwise :	Highest 106°	150	
	Lowest 100·2°	100	

Case 5. Febrile.	Highest 102·6°	140	44
	Lowest 100°	122	30

Case 6. "At first a high temperature, but this was not the case latterly."

Case 7. No note.

	Temp.	Pulse.	Resp.
Case 8. Febrile.	Highest 102°	120	38
	Lowest 99·5°	104	24

Case 9. On the whole febrile throughout, but irregular; thus, on admission it was 98·4°, and on three occasions between 98·4° and 99°. These low temperatures were probably accounted for by hæmorrhage or were the immediate effect of the operation. In the last twenty-four hours it was 97·4° on two occasions. The pulse was always quick.

	Temp.	Pulse.
Highest	103°	146
Lowest	97·4°	108

Case 11. Febrile on the whole, but the last two days the temperature was subnormal, and at the time of death 111° in the rectum; otherwise:

	Temp.	Pulse.
Highest	103·6°	104
Lowest	99°	84

Case 12. Febrile, but at times very low, thus on four occasions it varied between 97° and 97·8°, and on fifteen between 98° and 99°. The evening temperature was sometimes lower than that in the morning.

	Temp.	Pulse.	Resp.
Highest	103·6°	134	35
Lowest	97·4°	100	24

Case 13. Said to have never been febrile, but it was not regularly taken.

No doubt the range and variation of these factors can be best estimated by reference to the charts, and unfortunately these are not as complete as could be wished. It appears, however, to be clear that with the doubtful exception of Case 13 the temperature was always irregularly febrile, and the pulse and respirations, when noted, were quickened. The variation was extremely irregular, and rarely the evening was lower than the morning record; further, it will be seen that some very high and some very low temperatures were noted.

I have now gone through in detail what have seemed to me to be the important and prominent features of the cases I have recorded. I fear it will be considered in very wearisome detail, but I have thought that the abstracts might be of use to those who may consider the cases worth studying and do not care to wade through the detailed histories. The points hitherto touched upon represent the condition of the patient as it will probably be presented clinically; it only remains now to consider the etiology of this variety of hepatic suppuration, and to ascertain how far the symptoms due to each particular cause either have assisted, or will be likely to assist in diagnosis, and then to make a general *résumé* of the whole.

The causes, as far as ascertainable, in the cases I have cited were as follows:

Cases 1, 2, and 3. Indeterminate. It may be objected that there was some ulceration of the cæcal appendix in Case 3, but it was very slight indeed, and did not satisfy Dr. Goodhart's mind as to its being a sufficient cause; I think it certain that any one who saw the specimen would have been of the same opinion.

Case 4. Duodenal ulcer.

Cases 5, 8, and 10. Ulceration of cæcal appendix.

Case 6. Ulceration of cervix uteri.

Case 7. Gastric ulcer.

Case 9. Gangrenous dysenteric ulceration.

Cases 11, 12, and 13. All resembled one another in being due to prolonged obstruction to the outflow of bile, but in 11 and 12 this was due to old inflammation about the gall-bladder sequential to calculi, and in 13 to the blocking of the

common duct by a single nodule of encephaloid cancer, which was of small size and the only one in the body.

I have already indicated at the commencement of these remarks, how it seems probable that the different causes may be grouped, and I will simply add here a list of the various primary lesions that have been discovered in other cases than those cited in my paper, all of which I believe are susceptible of the same classification. Dr. Hilton Fagge¹ writes: "In addition to ulcerative affections of the stomach or bowels, suppuration of the spleen, suppuration of the mesenteric glands, and the penetration of one of the veins which go to form the portal trunk by foreign bodies. In a case quoted by Frerichs a fish bone had entered the inferior mesenteric vein. Again, inflammation of the capsule that encloses a hydatid cyst of the liver not rarely sets up suppuration along the portal canals within the organ, which in this case, however, seems to depend upon inflammation of the branches of the bile-ducts, rather than of those of the portal vein."

Dr. Murchison,² in addition to cases due to causes already mentioned, gives one due to softening tubercle in the mediastinal glands, at least this was the only lesion found besides the hepatic, another to ulceration of the cystic duct by gall-stones, and another to cancerous ulceration of the stomach. He also relates several cases of hydatids opening into the biliary passages, all of which appear to confirm the above quoted statement of Dr. Fagge as to the site of the suppuration. Drs. Wilks and Moxon³ state that "the disease which gives rise to such suppuration may be in the alimentary canal, gall-bladder, or spleen; the portal suppuration arising through an extension of the inflammation *along the wall of the implicated branch*. * *

* * It is sometimes very difficult to determine whether suppuration of the vein may not be primary, and the abscess in the field of origin of the vein secondary, and caused by it." They do not discard the embolic theory as one cause of pyæmic suppuration, but point out (p. 442) that the nature of the process set up in the liver will depend upon the condition of the lesion furnishing the emboli. Here I may call attention to

¹ "Diseases of the Liver," 'Guy's Hospital Reports,' 1875.

² 'Diseases of the Liver,' 2nd ed., pp. 169, *et seq.*

³ 'Pathology,' p. 445.

Case 9 for two reasons, firstly, that the gangrenous nature of the patches in the liver corresponded with the condition of the colon; and secondly, because of the apparently very rare occurrence of this state of the liver.¹

One case of pylephlebitis cited in this work² was due to suppuration in the submucous tissue of the rectum. This case is related in the post-mortem records of Guy's Hospital for 1870, and further causes collected from the same source have been:—Ulcerated stricture of rectum (1860); suppuration of ovary communicating with rectum (1874); cancer of pancreas causing obstruction of bile-duct (1875); and finally, tropical abscess which, in one case gave rise to multiple pyæmic abscesses around it (1876).

This list does not profess to be complete, except for the years mentioned, and further, as I have already stated, it does not include all cases of suppuration of the liver, the purpose of my paper being to examine into the clinical aspect of multiple small suppurations, which *do not give rise to the formation of obvious tumour*; and moreover, to take only those cases in which the primary lesion was not of a nature to overshadow by its symptoms those due to the hepatic affection. The inquiry was suggested by the occurrence of Case 3 during the time I was a clinical assistant. The remarkable nature of that case excited my attention, and I was fortunate enough to meet with five examples subsequently, viz. Nos. 8, 9, 11, 12, and 13.

I propose now to examine how far the symptoms of the primary lesion may from an examination of the histories be expected to assist in the diagnosis.

In Cases 1, 2, and 3, no primary lesion was ascertained.

In Case 4, the pains in the side for three months before admission, and the vomiting of blood one week before, were apparently referable to the duodenal ulcer.

In Case 5 there is nothing in the report, which suggests that there was a clear history of perityphlitis.

In Case 6 there was a history of abortion, but there was also one of biliary colic; the hepatic suppuration was due to the former, but clinically probably the latter would have overshadowed the actual cause.

¹ Murchison's 'Diseases of the Liver,' p. 120.

² Wilks and Moxon, 'Pathology,' p. 443.

In Case 7 there is no history of the gastric ulcer having caused any symptoms, and it is, of course, a familiar fact that this lesion may give no evidence of its presence.

Case 8 gave a history of a sudden onset of acute peritonitis.

Case 9 came under my own care. We had a history of the passage of "blood and slime," but all the stools passed in hospital were typhoid in character, and the subsequent hæmorrhage from the bowel corresponded to the time at which this complication usually occurs in enterica.

In Case 10 there was a history of biliary colic, and a doubtful one in Case 12.

In Case 13 there was no previous history to guide, but it was obvious from the deep olive-green hue of the skin that there was some long-continued cause of obstruction to the duct.

It appears from the foregoing that it may be taken as a general rule that the previous history will certainly fail to help in some cases, and will often be very vague; again, in others it will be of the utmost use. If now reference be made to the detailed examination of the clinical aspect, here again it is found that each particular symptom may be absent in any one case, although there was no case in which all those detailed were absent. First in order of frequency and importance appears to be a febrile temperature. Of Cases 1 and 2 there are no details as to this point, and Case 13, in the absence of a regular and systematic record, must go for nothing on this point. Murchison¹ has, indeed, placed on record a case of multiple pyæmic abscesses of the liver with no pyrexia. A febrile temperature will, however, doubtless be present in the great majority of cases. Given the fact of elevation of temperature, is there anything to call attention to the abdominal cavity? I think the conclusions arrived at under the headings, "Aspect of patient," "Appearance of abdomen," "Abdominal pain and tenderness," and "Hepatic enlargement," will answer very strongly in the affirmative: in the majority of the cases there will be such indications. Here I may perhaps allude again to the peritoneal rub which occurred in Case 3. Wasting was prominent throughout. In several cases there was extreme apathy. A dry, brown

¹ 'Disease of the Liver,' p. 175.

tongue was also common; sweating, rigors, vomiting, and constipation, or diarrhoea, all appear to be less constant symptoms. Finally, I should like to refer somewhat at length to the question of jaundice; it was only present in a third of the cases cited, and in two others there was a sallow tinge. Dr. Hilton Fagge¹ states that jaundice and a febrile temperature "almost always" accompany pylephlebitis. This does not agree with the results deduced from my thirteen cases, but this probably arises from the fact that they are so few. In the cases cited by Dr. Murchison,² it was present in four out of seven cases, one of which was the case without elevation of temperature already mentioned.

I do not think it necessary to do more than allude to the febrile jaundice accompanying the specific fevers, acute tuberculosis of the liver, and sometimes pneumonia, although perhaps Case 9, from its close resemblance to enterica, suggests that the point is one of importance. Murchison³ says that the liver and spleen are enlarged in 30·3 per cent. of cases of enterica, "but it is rare that either organ is the seat of pain or tenderness."

It appears, therefore, as the result of the examination of these thirteen cases, that there are no signs or symptoms pathognomonic of the condition of the liver under consideration, and that both fever and jaundice may be absent. Further, in connection with this point, it is important to bear in mind that acute yellow atrophy of the liver *may* be accompanied by fever, although in the majority of cases the temperature is normal or even subnormal. Thus Dr. Fagge¹ gives one temperature of 101·6°, and in a case admitted under Dr. Wilks into Guy's Hospital March 29th, 1878, the range of temperature was as follows:—April 8th, morning 99°, evening 100·4°; 9th, morning 101·8°, twelve noon 102·2°, 2.15 p.m. 104°, 6.15 p.m. 104·6°. She died at 8 p.m. Such a case might be confounded with pylephlebitis. Conversely in Case 8 some crystals, resembling leucin and tyrosin, are described as occurring in the urine; there was, however, *excess* of urea.

The diagnosis of a particular case will be manifest or obscure

¹ 'Guy's Hospital Reports,' 1875, "Diseases of the Liver."

² 'Diseases of the Liver.'

³ 'Continued Fevers,' p. 148.

in proportion to the presence or absence of signs or symptoms drawing attention to the general condition of the patient, the state of the liver, and the existence of certain lesions capable of producing portal infection, or obstruction of the bile-duct. A typical case would be probably one presenting all the symptoms detailed above under the twelve headings, together with a history of some possible primary cause of hepatic suppuration; but such a case will be rare, and there will be all gradations down to complete obscurity. This is, of course, what every one knew before; but I venture to urge as my excuse for publishing the paper, chiefly the undoubted intrinsic interest of the cases themselves, and the feeling that negative results are useful, if only to promote further inquiry. Most important of all, perhaps, is the suggestion thrown out by Case 10 of the possibility of recovery in this form of suppuration of the liver. I have notes of another case, which I am not able to publish, also pointing in this direction, and possibly also the following extract from the autopsy of Case 3 may indicate the same:—"Many of the points, however, were peculiarly white and tough for small suppurations, and were without any zone of congestion around." This, however, was an acute case. Dr. Wilks' remarks on the pathology of Case 1 are also very suggestive.

Finally, Murchison¹ gives a case which may be read in connection with this matter. I will simply copy part of the heading, together with an extract from the report of the autopsy. "Gall-stones causing Ulceration of Cystic Duct—Pyæmic Hepatitis." . . . "Liver studded with numerous inflammatory deposits, up to the size of a cherry, most of them consisting of a firm, translucent, greyish material, which in some cases was softening into an opaque fluid, like pus. The former material was made up of branched fibre-cells, and the yellow fluid of oil globules and compound granular bodies, but no true pus corpuscles." Might this possibly be read in the opposite direction, viz. that the fibroid material was curative, not degenerating?

¹ 'Diseases of the Liver,' p. 176.

CASE 1.—*Suppurative inflammation of the portal vein ; acute peritonitis.*

(Reported by Mr. S. PRALL.)

S. A—, æt. 35, a labourer, living at Brixton, was admitted on February 11th, 1857, under the care of Dr. Owen Rees.

Previous history.—He was of temperate habits, and prior to this illness had always enjoyed good health.

Present illness.—Commenced, as well as he was able to state, some days before Christmas with shivering, abdominal pain and tenderness, and vomiting.

At the end of a fortnight the last-mentioned symptom ceased, but again supervened two days before his admission. Five weeks from the onset the abdomen began to swell. His bowels had been constipated throughout, never acting without medicine.

He had no cough, neither had he brought up any blood by vomiting or otherwise. The reappearance of the sickness was accompanied by severe shivering, but he had suffered from this more or less during the whole of his illness. He was obliged to give up work very early in the attack, and had been attended by the parish doctor throughout.

Condition on admission.—He was wasted, with sunken eyes. His face was yellowish and sallow, but the conjunctivæ were pale. He was exceedingly low, and did not speak above a whisper. The respiration was hurried, but there was neither cough nor expectoration. The pulse was extremely feeble. The abdomen was distended with fluid, and was tender on pressure. He was constantly vomiting a dark greenish-coloured fluid.

He was ordered poultices to the abdomen, enema Olei Ricini, two ounces of wine, and whatever nourishment he could take.

Progress of the case.—February 12th.—His skin was cool, his pulse exceedingly feeble. His respiration was rather quicker than normal. The vomiting continued. There had been a slight action of the bowels, the evacuation being clay-coloured.

13th.—The vomiting continued. The bowels acted once after an enema. The pulse became imperceptible, and he died

in the course of the night. The report adds "without the symptoms throwing much light upon the nature of the complaint."

The autopsy was made by Dr. Wilks two and a half days after death.

There was commencing decomposition. Rigor mortis imperfect.

Body rather wasted. Skin slightly yellow, but partaking more of the sallowness produced by a miasmatic influence than that of jaundice. Head not examined.

Old adhesion of base of left lung to diaphragm.

Lungs: Bronchi healthy.

Heart: Weight 10 oz. This and the pericardium healthy.

Abdomen: acute and chronic peritonitis. There were three or four pints of serum in the abdomen, much lymph floating in this, and the intestines were united together. These could be separated without much difficulty at the lower part of the abdomen, but at the upper part the inflammation was older and the adhesions more firm; thus the omentum, which was firmly adherent to the parietes in front, was also closely attached to the colon beneath. The colon, stomach, and liver were all rather firmly adherent to each other, and purulent matter was found confined in the spaces between them. A large collection of yellow purulent matter was confined by adhesions between the spleen and the diaphragm.

Liver: Upon cutting through the portal vessels in separating the liver from the duodenum a large quantity of pus escaped from Glisson's capsule, and from the portal vein. Behind the pancreas was a circumscribed abscess holding about $\frac{1}{2}$ oz. of pus. The pancreas itself was healthy, as was also the duct, and the splenic vein running behind it was quite unaffected. The mesenteric vein, too, when cut through was quite healthy. The whole of the suppuration thus appeared confined to the lesser omentum and to the portal vein.

Upon removing the liver it was difficult to say, at first sight, what structures were principally affected, from there being so much suppuration about the vessels, and also from the fact that more than one part had been invaded by the severity of the inflammation. It appeared, however, after careful dissection that the hepatic artery was quite healthy throughout. Also that the hepatic ducts were not the primary or principal seat

of disease, for although in parts of the liver, where considerable abscesses had formed, the smaller ducts were found involved, and contained purulent fluid, yet the main branches of the hepatic ducts were unaffected. Their walls presented no appearance of inflammatory exudation, and although the tubes contained much yellow tenacious bile yet they were not at all distended. Whatever disease of the smaller ducts existed, therefore, was no doubt secondary, and caused by the general structures of the liver being involved in the suppurative process.

The portal veins were extremely diseased throughout, being in a state of intense suppurative inflammation. They were all much dilated, quite distended, with a pure pus of a yellowish colour. Nearly the whole of the liver was thus affected. A section in any part which cut through the portal vessels showed a purulent fluid running from the vein. Not merely did they contain pus, but their coats had been subject to a severe inflammation. Thus the walls were softened and thick, and in parts detached from the hepatic tissue around. In the large trunks there were distinct patches of lymph adherent to the coats, and in the trunk itself of the vena portæ there was a firm layer of pink-coloured lymph closely adherent to the wall.

In some parts of the right lobe the suppuration had advanced as far as the capillaries and the secreting system, so that a section of the organ displayed the lobules distinctly mapped out, the lobules themselves being of a yellowish colour from the suppuration going on within them, and the hepatic vein still visible as a red spot in the middle. In some few places these suppurating lobules had run together, and having become much softened would in a very short time have formed distinct abscesses. A most remarkable fact was that one of the larger trunks of the portal vein was quite unaffected, yet when traced upwards its branches were found distended with pus. It appeared from this that the inflammatory process had been primarily and mainly in Glisson's capsule, and that the tubes had been afterwards involved, and perhaps from the periphery towards the trunks.

That the inflammation had been primarily in Glisson's capsule was shown also in the fact of there being an exudation of

lymph or fibrinous material of firm consistence and old standing in the same structure. Thus, in various sections of the liver where the portal veins were exposed, a whitish material of different consistences was seen, in one place firm, as in cirrhosis, in another soft and cheesy. This appearance was, however, but slight and very limited. That the inflammation was also of some standing, was shown by the great thickening of the coat of the portal vein which formed part of the walls of the abscess beneath the pancreas.

The coat of the vena portæ was three times thicker on this side than it was on the other. The cystic and hepatic ducts were healthy.

Gall-bladder full of ordinary bile. No gall-stones.

Weight of liver 4lb. 3½ oz.

Weight of spleen 11½ oz.; its peritoneal surface inflamed; otherwise healthy.

Pancreas and its duct healthy, the under surface at one spot of a dark colour where in contact with the suppurating parts. The tissue beneath slightly softened, but not otherwise diseased.

Supra-renal capsules healthy.

Kidneys healthy, weighing 9½ oz.

Stomach was probably healthy, the mucous membrane was of a greyish colour and covered with white spots. This, however, was no doubt due to decomposition.

Intestines healthy, carefully examined, and no trace of ulceration discoverable.

Remarks by Dr. Wilks.—This case is remarkable for its rarity, its obscurity, and its pathology. The more chronic affection of Glisson's capsule and the portal system of vessels, as in cirrhosis, is displayed in the production of a hard, fibrous exudation, while the more acute, where suppuration is present, generally arises from disease in some part supplied by the portal vein and is of a phlebitic character. It is a question how far this case partakes of one or the other of these forms of disease, whether the inflammation of the vein be altogether secondary to some previous abscess (of unknown origin) in the neighbourhood of the pancreas, or whether it has been an altogether primary inflammation of Glisson's capsule, but for some undiscovered reason acute and suppurative.

CASE 2.—*Suppurative phlebitis of liver.*

J. C— was admitted on January 27th, 1864, under the care of Dr. Wilks.

The patient, a male, æt. 48, came from Edmonton. He had been ailing since Christmas time, with abdominal pains, diarrhœa, and occasional sickness. He got thin and was obliged to keep his bed.

On admission.—He was very ill. He had no acute symptoms but was thin, pale, and looked ill.

The abdomen was somewhat tumid and tender. There was more or less diarrhœa.

He remained much in the same state, but without any more marked symptoms, until about two days before death when sickness came on and he rapidly sank.

Autopsy.—Body wasted; head not examined. There was recent pleurisy on the right side, at the lower part, evidently proceeding from the diaphragm. The base of the lung was adherent, and there was a little lymph on the surface of the lower lobe. The lungs were healthy, and there was no appearance suggesting pyæmia.

The liver was adherent to the diaphragm above, and to the stomach below. The adhesions were composed of recent lymph, so that they were readily separated.

On exposing the liver, numerous spots of suppuration were seen on the surface, and these would have burst had it not have been for the adhesions.

On cutting into the organ, it was seen to be studded by numerous small abscesses. These were situated more especially towards the upper part of the right lobe. They were crowded together and not of large size; it was observed, however, that each one poured forth a large quantity of pus mixed with bile, showing that the matter was carried by a vessel from a distance. On more careful examination it was clear that the suppuration existed in the course of the portal system. The ducts were considerably distended.

CASE 3.—*Pylephlebitis; multiple abscesses of the liver; peritonitis.*

(Reported by Mr. C. J. SYMONDS.)

D. R—, æt. 26, a policeman, was admitted, on November 26th, 1875, under the care of Dr. Wilks.

Previous history.—Six years ago, whilst in Texas, had ague and cholera. He has had no return of the former since he has been in England. He has been in the force for five years, and has never been ill during that time. He has never had jaundice, nor biliary colic, nor hæmorrhoids. He has lately been on night duty over some cells whence there has been a rather bad smell. There has been no illness amongst his fellows lately.

Present illness.—The patient was in good health on Nov. 21st, and went on duty on the night of the 22nd feeling quite well, but in the course of it had pain and aching in the loins and right side. He went to bed at 6 a.m., as usual, on the 23rd, but was feeling “poorly” and aching all over, and on rising was unable to eat his breakfast, nor could he sleep at all during the day. In the evening he saw a doctor, who prescribed for him, and ordered port wine. He took a wine-glassful, but was immediately seized with abdominal pain, and vomited it. On the 24th the pain in the back still continued, and there was also some in the abdomen, especially after the wine. On the 25th he had pain in all his limbs, and remained in bed. On the 26th the abdominal pain diminished, but that in the loins and limbs became worse, and his joints felt stiff. The bowels acted twice on the 21st, and again on the 24th, but not since. He has had neither headache, nor sickness, nor cough, nor stitch in the side, nor abdominal swelling, nor urinary trouble, nor shivering, nor sweating. He says he has lost two stones in weight during the week.

Condition on admission.—He walked into the ward supported by one man. His eyes were sunken. He complained only of aching in all his limbs. The chest was well formed, filled well, and was normal to auscultation and percussion. His respiration was more thoracic than abdominal. There was no general tenderness nor distension of the abdomen, but pain was produced over the liver by pressing beneath the ribs, and there

was also slight tenderness below the spleen, but it was not great. Hepatic dulness commenced at the fifth rib, and extended a finger's breadth below the costal margin. *Over the region of the liver there was heard, with inspiration and expiration, a rub, made up of a few prolonged sounds.* This was not heard in passing over the lungs where the vesicular murmur was audible. The spleen could be felt hard and rounded below the ribs. The urine contained one fourth of albumen, but no casts nor sugar. The temperature, pulse, and respirations up to 10 a.m. of the 27th were as follows :

		Temp.		Pulse.		Resp.
November 26th, 7 P.M.	. .	104·7°	...	132	...	32
9 P.M.	. .	102·8°	...	160	...	34
„ 27th, 2 A.M.	. .	104·8°	...	160	...	28
5 A.M.	. .	103·8°	...	—	...	—
10 A.M.	. .	102·6°	...	140	...	36

Progress of the case.—November 27th.—The nurse said he had had scarcely any sleep. He looked very ill and anxious, his face was thin, and twitched frequently, his eyes were sunken with dark rings around them, the “*facies Hippocratica*” was well marked. His arms hung stiffly with the wrists drooping. He was unable to move his joints quickly, and when this was done for him, it caused pain, but after they had been moved several times he could easily use them himself. His knees were sometimes flexed, at others extended. There was no redness, nor puffiness about the joints. He gave his history excitedly, but always gave the same dates. He said he ached all over if he laid for a quarter of an hour in the same position, and he was constantly shifting about. His skin was hot and dry, and the extremities were warm. His tongue was dry, rough, and horny. His pulse was regular, but very weak and compressible.

1.15 p.m.—There was a reddish swelling on the left forearm, said to have been caused by a fall in attempting to reach the water-closet last night. The pulse was regular, very small, and could not be counted at the wrist. The temperature was 103·5°, the respirations 44.

4 p.m.—The tongue was dry, red, horny, and fissured. There was now slight pain and tenderness over the abdomen, especially in the region of the umbilicus. The joints were still

stiff, but none were swollen. He spoke excitedly, but rationally.

9 p.m.—He was sweating profusely. Wanted to get up and go out. Temp. 106.2° , pulse 144, resp. 40.

Midnight.—Was still sweating. He was more inclined to wander. There were no further local symptoms. Temp. 105.8° , pulse 172, scarcely perceptible at the wrist, resp. 36.

28th (2.45 a.m.).—He lay unconscious. Pupils were rather contracted; conjunctivæ were sensitive. The pulse was imperceptible at the wrist. Temp. 106.8° , pulse 192, resp. 30.

He remained unconscious. The nurse said he had three or four convulsions in which he trembled all over, and he died at 4 a.m.

The autopsy was made thirty-seven hours after death by Dr. Goodhart. I have quoted verbatim the record, with the heading as written by him, and have added that of the case of idiopathic pyæmia to which he refers, as elucidating the pathology of the one under consideration. The heading of the examination was as follows:

“Idiopathic pyæmia; suppurative phlebitis of the portal vein; diffuse suppuration in the liver; peritonitis.”

“A muscular man. Firm rigor. Much hypostasis. No trace of any scar or sore externally. I incised the muscles of the extremities in all directions, but without finding any local disease whatever. The veins were all healthy. The bones also. Joints all free from injection or pus. The axillary and inguinal glands on both sides were, however, twice to three times their normal size, and deeply congested, indicating some general disease.

“All the viscera were perfectly healthy, except the liver and cæcal appendix. The latter was *very*¹ superficially ulcerated, and the mucous membrane a little thick and injected. It did not appear, however, that it was sufficiently diseased to have led to the state of the liver. The liver shows slight peritonitis over its surface; it weighs 98 oz. The right lobe and part of the left are in a very remarkable state, studded over with minute points of suppuration, the size of a pin’s head. On section

¹ The italics are Dr. Goodhart’s.

these were found to be scattered over both the deep and superficial parts, but were more numerous towards the surface. In all in which the connection could be traced, the pus point was in a radicle of the portal vein. They were breaking down in some parts, and in two places I found a cavity the size of a filbert, with chocolate-coloured contents, from broken-down liver substance. Many of the points, however, were peculiarly white and tough for small suppurations, and were without any zone of congestion around. Tracing the portal vein towards the transverse fissure, the main branches of the right lobe were partially plugged by breaking down blood-clot. This state extended to the second and third subdivisions, then stopped, and the remainder of the vein was healthy for some distance till the more terminal part was approached. The left lobe had similar minute suppurations in it near to its left edge, and some small local clots were found adherent to the walls of branches of the portal vein.

“No cause for the suppuration could be detected, but whether or no it be believed to be secondary to some undiscovered cause of a primary phlebitis of the portal vein, I think the case should be considered in connection with that of idiopathic pyæmia occurring on November 26th, 1875, as suggesting some extensive factor leading to blood poisoning, and not any local suppuration as a primary source.”

Spleen weighs 23 oz., and is very pulpy.

The following is the case alluded to by Dr. Goodhart :

Idiopathic pyæmia.

J. W—, æt. 38, was admitted under the care of Dr. Pavy on November 19th, 1875.

Previous history.—No illness of any kind. He had been a great drinker, being in the habit of taking a gallon of beer a day.

Present illness.—Five days before admission he was flooded out of his house by high tides, and so got wet. Two days after this he was suddenly seized with pain in the muscles of both legs and arms. He became delirious on the second day of his seizure.

Condition on admission.—He is a strong, healthy-looking man. His intellect is clear. He has pain in the shoulders

and right knee, and the latter is red and swollen. There is general muscular tremor. His temperature is 104.5° , his pulse 124, and his respirations 28. In the evening, 11 p.m., he was sweating profusely, was delirious, and his tongue was furred. Temp. 103° , pulse 108, resp. 24.

Progress of the case.—November 20th.—The delirium has increased.

		Temp.		Pulse.		Resp.
M.	.	103.4°	...	112	...	—
E.	.	105.2°	...	132	...	28

21st.—Temp., morning 101.2° , evening 101.6° . Still wandering.

22nd.—

		Temp.		Pulse.		Resp.
M.	.	99.8°	...	—	...	—
E.	.	102°	...	118	...	38

23rd.—Both knee-joints swollen and red.

				Temp.		Pulse.
M.	.	.	.	103.6°	...	116
E.	.	.	.	103.8°	...	—

24th.—Is restless. There is pain in the elbows and wrists ; no swelling. Is sweating profusely. Temp. 104.8° .

25th.—There is much tremor. There is dusky red swelling of the left wrist. Abdomen somewhat distended and tympanitic, and apparently painful. No vomiting. His pulse hardly perceptible. Much subsultus tendinum. He is evidently dying of blood poisoning.

The blood was examined. A large number of white blood-cells of three sizes were found. He died at 1.45 p.m.

Autopsy, twenty-five hours after death, by Dr. Goodhart.

The body was muscular. There was a small scab over the metatarso-phalangeal joint of the right little finger, but with no redness nor suppuration around it.

Both lungs were full of blood.

The heart was quite healthy ; it weighed 13 oz.

The peritoneum was slightly injected. This was the only existing evidence of any inflammation.

The liver was fatty, weighing $84\frac{1}{2}$ oz.

The spleen weighed 13 oz. Its tissue was quite pulpy.

The kidneys were healthy.

The right shoulder contained pus, which extended down the bicipital groove. The left wrist was also full of pus.

Both knees were also similarly affected. On the right side the suppuration extended from the joint up the thigh, in the rectus and adductor muscles, nearly to the groin. There were localised deposits of pus in the substance of the muscles. The glands of both groins were swollen.

The examination was most minutely conducted, with the view of finding the primary source of the pyæmia, but without success. It would be tedious to write down the negative results of the scrutiny, but every possible source was sought for. The pus in the left knee was examined immediately after death. It contained some dumb-bells, much granular matter, and well-formed pus cells.

CASE 4.—Ulcer of the duodenum, obliterating the portal vein, destroying the hepatic and cystic ducts, and opening the hepatic artery; portal pyæmia; suppurative peritonitis; cystic ovary.

(Reported by Mr. S. V. INSTONE.)

S. T—, æt. 45, a married woman, was admitted on September 16th, 1875, under the care of Dr. Habershon.

Family history.—Phthisis on her father's side.

Previous history.—Had had four children, all of whom were dead. Had enjoyed good health up to twelve years prior to her present illness. She then had a fall from a railway carriage, which laid her up for some months, and after this she became subject to fits. She had rheumatic fever one year before admission, since which illness she had been always ailing more or less. There was also a probable history of abortion at the fourth month, about three months prior to her coming in.

Present illness, dating from the time of admission, commenced three months before with pain in the side, which had continued since. Three weeks before she had had cold shivers, lasting for about six hours; after this she continued to get worse. One week before she vomited nearly three pints of dark clotted blood.

Condition on admission.—Patient was emaciated, with sallow complexion, bright eyes, hot and dry skin. She complained

of sharp cutting pain in the right side. The heart was normal. The pulse 100, weak, compressible. The left chest expanded better than the right. The right side was resonant, and the breathing was puerile. On the left side there was some dulness and great tenderness to percussion at the apex, and bronchial breathing and bronchophony at the base in front. She had a short, dry, hacking cough. The tongue was covered with a creamy fur. The abdominal muscles were very tense. The hepatic dulness extended nearly to the level of the umbilicus. The urine was normal, its sp. gr. 1010. The catamenia regular. Temperature 100·4.°

Progress of the case.—She steadily went on to a fatal termination, which occurred on October 11th. I will state briefly the most noticeable of her symptoms, and will indicate the range of pulse and temperature in tabular form at the end. The treatment that was adopted consisted chiefly of opium and stimulants, and at the time of her vomiting blood she was ordered oil of turpentine, and fed with nutrient enemata.

Up to September 26th great pain in the right side is noted every day. On September 22nd she had pain in the right shoulder. From September 26th to October 2nd great pain in the right side is noted. On October 6th she complained of abdominal pain when she coughed. On October 9th and 10th pain in the right side is again recorded.

On September 18th she vomited dark clotted blood. On September 30th she had great nausea, and the next morning she was sick. On the morning of October 6th she vomited a pint and a half of fluid stained with dark blood, containing a few small clots; and later on in the day another pint of the same fluid. Again, on October 8th, she had nausea, but was not sick.

On September 19th the abdomen was very tender all over. Again on October 9th, it is noted that there was great abdominal distension and tenderness.

On September 18th she had slight diarrhœa. From this time up to September 26th there was complete constipation, but she was taking opium. On September 27th, 28th, and 29th, an action of the bowels is noted. On October 7th there was continued action from 2 o'clock in the day until evening. The fæces on this day are stated to be dark and pitchy. On

October 7th it is recorded that the tongue was slightly furred. On September 23rd fine crepitations are noted at the right apex, and on September 27th bronchial breathing at the same spot. On the night of October 7th she had several attacks of dyspnœa, and again, on October 9th and 10th, severe paroxysms are stated to have occurred from time to time. The following record probably relates to the morning :

			Temp.		Pulse.
September	17th	. . .	100·4°	...	100
"	18th	. . .	104°	...	130
"	19th	. . .	106°	...	140
"	20th	. . .	102°	...	125
"	21st	. . .	102°	...	120
"	22nd	. . .	103°	...	138
"	23rd	. . .	102·6°	...	120
"	24th	. . .	102°	...	120
"	26th	. . .	102°	...	120
"	27th	. . .	104°	...	120
"	28th	. . .	101°	...	120
"	30th	. . .	102°	...	115
October	1st	. . .	103·4°	...	116 very weak
"	2nd	. . .	101°	...	120
"	4th	. . .	101·4°	...	110
"	6th	. . .	102°	...	120
"	7th	. . .	98·4°	...	130
"	8th	. . .	101·4°	...	150
"	9th	. . .	102·2°	...	150
"	10th	. . .	100·2°	...	150

The autopsy was made eight hours after death by Dr. Goodhart. The record is quoted almost literatim, and the comments on the appearances presented are his.

The body was somewhat emaciated. There was some recent lymph at the back part of the right pleura. The lungs were healthy, except that the lower lobes of both were collapsed. The heart was healthy.

On opening the abdomen, a considerable quantity of pus was found bathing the intestinal coils. The liver, close to the round ligament, was found adherent to the abdominal wall, and a small abscess lay between the two. This had no doubt been originally situated within the hepatic substance, but had ruptured into the peritoneum. One inch beyond the pylorus was an ulcer in the duodenum. This had now only an edge at one part, elsewhere it was merely a ragged sloughing

cavity, occupying the whole of the portal fissure, the edges of which were adherent to the stomach, so that no communication existed with the peritoneum. The cavity had so eaten into the portal fissure that the hepatic and cystic ducts had quite disappeared. The gall-bladder, of which the mucous membrane was acutely inflamed, opened directly by its duct into the cavity. It contained a good quantity of healthy bile. The hepatic duct ended in the same manner, though apparently by some dilatation at the fissure, in which was collected some soft material, resembling inspissated bile, which further examination seemed to prove to consist of blood clot altered by bile.

The hepatic artery also opened into the floor of the cavity. The splenic vein was normal, its junction with the inferior mesenteric unaffected, and it also had a free communication with a gastric radicle of some size, which ran up on the left of the portal fissure, and entered the liver. The portal vein lay gaping in the floor of the ulcer, just above the pancreas; it was quite obstructed from the fissure to the back of the pancreas, where the abscess burrowed downwards. This vessel in the fissure contained emboli of some date, which were coloured with bile. The duodenal end of the common bile-duct lay with its mouth in the ulcer base.

The liver itself was studded throughout with small abscesses, which were most numerous in the left lobe. Why this lobe was more affected than the right could not be determined. "The abscesses, in the liver were, I think, in the radicles of the portal vein, but I could not quite decide whether this was so, or whether they were situated in dilated ducts."

The stomach was mammillated to a remarkable degree, studded with small granulations, and its mucous membrane looking like a piece of dried leather.

The intestines contained but very little, and that was slate-coloured; but bile was distinctly present in the faecal contents of the rectum. The kidneys were healthy. The spleen rather swollen, weighing 12 oz.

A quantity of pus had collected in the pelvis. The Fallopian tubes were matted to the ovary. The left formed one large cyst, nearly the size of the foetal head, full of porter-coloured fluid. The ovary itself was converted into three cysts, full of dirty brick-coloured, softening, *ante-mortem* clot; and the other

ovary was a mass of similar clot. "It was a question whether these were corpora lutea or not. I think not. The condition probably resulted from congestion consequent upon the peritonitis." The uterus was healthy.

CASE 5.¹—*Sloughing and ulceration of the cæcum; secondary abscess underneath the peritoneum; pylephlebitis; suppuration of the liver.*

(Reported by Mr. T. F. PEDLEY.)

G. D—, æt. 23, a married woman, was admitted into Addison Ward, on December 29th, 1875, under the care of Dr. Habershon.

Previous history.—Had enjoyed good health since childhood. She had a miscarriage four months ago, but completely recovered from it.

History of present illness.—It commenced three weeks ago with severe pain down the back, vomiting, and diarrhœa. She did not remember shivering.

Condition on admission.—The patient was much wasted; she lay on her back in bed, and moved with great difficulty. She was constantly moaning with pain, and was in a sleepy, dreamy condition, answering questions with difficulty, and in monosyllables. There was a pained expression on her face, which was flushed. Her eyes were bright, the pupils normal. The skin was hot and dry.

The tongue was dry, brown on the dorsum, pale at the edges. The teeth and gums were covered with sordes. She could only take fluids, of which she consumed large quantities. The bowels were relaxed; the stools being liquid and yellow. Hepatic dulness was much increased, and the region was tender on pressure. The splenic dulness was normal.

She had an occasional cough. Her breath had a peculiar sweetish odour. Respiration was free and thoracic, but short and quick. The lungs, with the exception of some fine crepitations at both bases, were normal.

The præcordial dulness was decreased in area from below. The heart's apex beat in the fourth intercostal space, two

¹ This case was recorded by Dr. Habershon in a paper on the "Diagnosis of Disease of the Cæcum," in the 'Guy's Hospital Reports,' vol. xxii, 1877, p. 290.

inches from the sternum. The cardiac sounds were normal. The pulse was small, regular, and compressible, 140.

The urine had to be drawn off. It was of normal colour, of sp. gr. 1010, and contained a slight trace of albumen. The chlorides were deficient.

Temp. 102·6°, pulse 140, resp. 30.

She was ordered Mist. Ammoniae Acetat. ℥j, sextis horis, and a draught containing fifteen grains of chloral every night. She was also placed on milk diet.

Progress of the case.—December 30th.—Patient lay semi-comatose. She said she was in pain all over the body. Temp. 102·2°, pulse 134, resp. 42. She was ordered—

Mist. Senegae ℥j, 4tis horis;

Pil. Doveri gr. v, 4tis horis;

Brandy ℥iv; beef-ten and eggs.

31st.—Last night she was very ill. She had continuous diarrhoea for six hours, and much retching and vomiting. She was now shivering, and complained of cold; and great pain all over her. Temp. 100°, pulse 130, resp. 44. She was ordered last evening—

Træ. Opii, ℥xv,

Decocti Amyli ℥ij. For an enema.

And the following mixture every four hours—

℞ Sp. Ammon. co. ℥xx,

Sp. Vini Gallici ℥ij,

Aquam ad ℥ss, every half hour.

January 1st.—She lay semi-comatose, with her eyes half open. She moaned, and said she was in great pain. Temp. 102·2°, pulse 122, resp. 44.

About twelve o'clock the diarrhoea again commenced, and Mist. Hæmatoxyli co. ℥j was administered at once.

4 p.m.—She complained of great pain in the cardiac region, threw her arms about, and said she was dying. The pulse was 140, weak and fluttering. She was troubled with flatulent eructations, and had been very sick in the afternoon. The fæces had the appearance of pea soup.

6 p.m.—She died, without convulsions.

The autopsy was made forty-three hours after death by Dr. Goodhart. The remarks in the course of the report are by him.

The body was sparsely nourished. The brain was healthy. The lungs were œdematous. The heart was healthy.

There was early peritonitis in the right hypochondriac region.

The stomach and small intestines were healthy. The cæcum and ascending colon lay in front of a considerable abscess which extended from the cæcum upwards behind the bowel and peritoneum as high as half way up the posterior surface of the right kidney. The cavity had evidently formed round the appendix cæci, which opened directly into the abscess; apparently about half of the appendix was wanting, and there was merely a stump now left. A second opening had formed about one and a half inches above the ileo-cæcal valve; this had sloughy edges, and was about one third of an inch in diameter. With these exceptions the cæcum and ileum were healthy.

The mesenteric veins exuded a copious amount of pus when cut across, especially those from the right colic region, and there appeared to have been a *diffuse* phlebitis in these vessels. The portal vein was plugged behind the pancreas and in the portal fissure by a firm cylindrical clot, which was smoothly channelled in its centre, and had evidently formed by lamination on the vein wall. There was no doubt that it had allowed the passage of blood, and this probably explained the absence of ascites. The main branches of the vein traced towards the liver were full of grumous pus and blood clot, and throughout the organ a quantity of pus was seen to exude from the branches of the vessel when cut across. The right lobe of the liver, near the lower surface, was riddled by sloughy abscesses, many of which had united to form one mass. These had extended to the surface, a grey slough was just breaking through, and an early peritonitis had been set up around it.

The tissues in the portal fissure were swollen, and much altered by inflammatory deposit and distended veins passing along with the hepatic duct. There was no evident blocking of the main ducts, but the gall-bladder only contained mucus. The organ weighed 73 oz.

The spleen weighed $14\frac{1}{2}$ oz., and was soft. Its vein looked healthy. The size was doubtless partly explicable by the obstruction, at any rate to some extent, to the return of blood by the portal vein.

The kidneys were healthy.

CASE 6.—*Gummatous liver; old inflammation about the portal fissure; ulceration of cervix uteri after delivery; plugging of uterine veins; pylephlebitis; abscesses in lung.*

I have unfortunately been unable to obtain the detailed history of the succeeding case, as it does not appear amongst the clinical records. This is less to be regretted from the point of view of my paper, as the suppuration of the portal vein was complicated with general pyæmia and syphilitic disease of the liver. I have thought it, however, best to include this case, as it is at least interesting, on the score of causation.

The abstract of the history I have given is that which heads the post-mortem record.

A. M—, æt. 27, was admitted on July 20th, 1876, under the care of Dr. Wilks. The patient was a married woman, and a bookfolder. Prior to her marriage she had had no serious illness, but a fortnight after she was seized with an attack of biliary colic, during which she passed several gall-stones. Six months afterwards she had a second such seizure, during which her skin became very dark, and her feet and abdomen were swollen. From this she recovered completely. Two months before admission, whilst expecting her confinement, she was taken with severe rigors, vomiting, and jaundice. On the second day she was delivered of a stillborn fœtus, which, according to her doctor, had been dead for some days. This was followed by fetid discharge for one week. Since this she has had diarrhœa at intervals. Two weeks before coming into the hospital her feet, legs, and abdomen began to swell, and she suffered occasionally from rigors.

On admission there was much ascites. She also had diarrhœa, and at first a high temperature, but this was not the case latterly.

She died gradually exhausted on August 2nd.

The autopsy was made by Dr. Goodhart twelve hours and a half after death. The remarks at the end are by him.

The body was spare. There were some cicatrices about the labia minora, but none elsewhere.

There was slight œdema of the legs.

The cranial bones and brain were healthy.

The cervical glands were normal.

There was a small amount of lymph on both pleuræ, and several small abscesses on the posterior aspect of the bases of both lungs.

The larynx, trachea, bronchial tubes, and mediastinum were all healthy.

The heart was normal.

The femoral veins were healthy.

There was much ascites. In Douglas's pouch was a local collection of pus shut off by adhesions from the peritoneal cavity. Old and tough adhesions existed about the portal fissure, matting all the parts together. These were evidently caused by a peritonitis of much earlier date.

The large and small intestines were lardaceous.

The liver weighed 67 oz. Its tissue was of a light fawn colour; in some parts fatty, in others congested. Running from the fissure for the gall-bladder in various directions was a deep irregular scar, which fissured and contracted the right lobe deeply, so that it was quite small. On section of the liver at this spot a large tough, grey, fibrous gumma was disclosed. On its deep surface it abutted on the portal fissure, rendering it possible that the adhesions at that spot arose from this cause. The ducts of the liver, the common duct, and the portal vein were patulous. The common duct, however, was large, and looked as though it had been dilated by the passage of a calculus; this was particularly suggested at the duodenal end. Throughout the liver large branches of the portal vein were full of pus, and large tracts of the hepatic substance were infiltrated with pus. Some of the suppurating veins passed close to the gumma, but the latter was nowhere breaking down, and therefore it is probable that the two conditions were independent.

The kidneys were healthy.

The entire cervix uteri was ulcerated and ragged looking, and its tissue was softened. Around this part the veins were plugged by ante-mortem coagula, none of which, however, were in a softened condition.

The veins in the uterine wall and broad ligament, and those of the ovarian plexus were healthy.

The ovaries were normal.

"I believe the course of events to have been this:—A large

gumma had formed in the liver; this had led to fibrous thickening in the portal canals, to inspissation of bile in the gall-bladder, and calculi had produced her earlier symptoms. Those of a later date were due to ulceration of the cervix uteri after labour, causing local plugging of veins, abscess behind the uterus from local peritonitis, embolism of small branches of the portal vein, suppuration of these, and portal and general pyæmia."

CASE 7.—Perforating ulcer of stomach; hepatic abscesses; rupture of one into the peritoneum; peritonitis; reduced hernia.

I have included this case in the series entirely on the score of causation. The clinical history was that of acute peritonitis. This was found post-mortem to be due to the rupture of a small hepatic abscess. The portal affection was so limited in its extent, that if it produced any symptoms at all, these must have been completely overshadowed by those due to the peritonitis. The case was rendered additionally obscure during life by the presence of a hernia, and indeed it was to the supposed rupture of this by taxis that the symptoms were attributed.

The patient was a man, æt. 23, who was admitted under the care of Mr. Bryant on October 18th, 1873.

At the autopsy which was made by Dr. Hilton Fagge, it was found that there was acute peritonitis; all the viscera being coated with a thick layer of lymph, which was in some places opaque and semi-purulent.

The hernia had taken no part in the causation of this, and the case seemed obscure, when it was discovered that there was an abscess, the size of a walnut or larger, in the edge of the left lobe of the liver, and that this had given way on its under surface. Around the main abscess were some small points of suppuration.

On opening the stomach, an oval ulcer with rounded edges was found near the lesser curvature. This viscus was adherent to the liver, and the ulcer had completely destroyed its coats, so that the base was formed by white tissue covering denuded

hepatic substance. A small fistulous opening led into a ragged cavity, the size of a large marble, in the left lobe of the liver. Around this was some suppuration along a portal canal, and no doubt it was by the transmission of the morbid material along some of the vessels from this focus that the suppuration in the edge of the left lobe had been set up; this latter was about two inches distant. The rest of the liver was healthy, and the gall-bladder contained a little yellow bile.

CASE 8.—Perforation of cæcal appendix; peritonitis; pylophlebitis; multiple abscesses of liver.

(Reported by Mr. E. B. GRAINGER.)

A. M—, æt. 15, plasterer's boy, was admitted under the care of Dr. Hilton Fagge, on June 1st, 1878.

Previous history.—No serious illness before. He had, however, frequently suffered from griping pains in the abdomen, from which he would recover in a few hours. His mother attributed this to improper food and drink of which she said he was very fond.

Present illness.—Eleven days before admission he came home feeling "poorly," and complaining of pain in the back. He said he had fallen and hurt himself whilst at play.

Nine days ago he returned from work at 10 a.m. doubled up with pain in the abdomen. He had been suffering from this since 6 a.m. and had been sick.

He was put to bed and prescribed for by a doctor, and has kept to it ever since. His symptoms have been headache, great abdominal pain, and vomiting. His face has been flushed and he has been hot and feverish the whole time. The bowels did not act for the first three days of his illness, and have been moved only three times prior to his admission. The day before he came into the hospital he passed no urine. On this day his mother found on his shirt a quantity of "matter" which "looked as though it came from a gathering." Jaundice also supervened on the same day.

Condition on admission.—He lay in bed on his right side with his knees drawn up. His face flushed and he looked ill and in pain. The conjunctivæ were deeply jaundiced; his skin hot, and dry. His lips were dry, and his tongue was covered with

a white fur. He felt sick. There was great tenderness over the whole abdomen, which was very hard with rigid recti muscles. The liver dulness extended from the sixth space to one inch below the margin of the ribs. Percussion caused great pain. The most tender spot was just below the ribs a little internal to the mammary line. Dr. Fagge in the evening thought he detected fluctuation in the right hypochondrium. The heart and lungs were normal. He had a hoarse incomplete sort of cough, but no expectoration. On the morning of admission he passed a large quantity of urine of the colour of porter. His urine had been high-coloured throughout his illness but never as dark as on this occasion. His temperature was 102° , his pulse 108, small, and hard, his respirations 28. He was ordered half a grain of opium every three hours, and hot fomentations to the abdomen.

Progress of the case.—June 2nd.—The abdomen was still tumid. The hepatic dulness did not now extend below the ribs. He passed his fæces under him this morning. They were fairly consistent and of a dirty-white colour. No water had been passed since the morning of the preceding day, so that the bladder was emptied by a catheter. The urine drawn off measured twelve ounces. It was of the colour of very strong tea, and gave the reaction for bile-pigment with nitric acid. It contained no albumen. The specific gravity was 1025. When evaporated it showed acicular and small violet hexagonal crystals.

3rd.—He was under the influence of opium, but otherwise in the same condition. He had not vomited since admission. He passed a motion in bed, and seventeen ounces of urine were drawn off by the catheter. The characters were the same as on the preceding day.

			Temp.		Pulse.		Resp.
A.M.	.	.	100.6°	...	108	...	24
P.M.	.	.	100°	...	104	...	24

4th.—The tongue was covered with a creamy-white fur. The bowels acted as yesterday, the fæces being clay-coloured. A pint of urine was drawn off by the catheter. His general condition was the same as on the preceding day.

			Temp.		Pulse.		Resp.
A.M.	.	.	100°	...	112	...	24
P.M.	.	.	101.3°	...	114	...	26

5th.—His abdomen was less tumid and the pain in it much diminished. There was comparative dulness on the right side for one inch below the ribs. Two pints of urine were drawn off, which was less dark in colour.

			Temp.		Pulse.		Resp.
A.M.	.	.	99.5°	...	120	...	28
P.M.	.	.	100°	...	108	...	27

6th.—The tongue was covered with a white fur. Soft mucous râles were audible with expiration over the whole front of the right chest, and the right base was dull behind; the respiratory murmur was less distinct, but there were no crepitations over this area. The urine measured two pints eight ounces; it was much less dark in colour. It was of specific gravity 1025, showed bile, but no albumen. "With hypobromous test it showed an excess of urea."

7th.—He seemed much worse. His skin was hot and perspiring, and his breathing was rapid and shallow. There had been no abatement of the jaundice since admission. Mucous râles were audible over the whole chest in front. He spat up white frothy mucus. The liver dulness extended one and a half inches below the costal margin. He did not complain of pain. He was with difficulty made to answer questions.

			Temp.		Pulse.		Resp.
A.M.	.	.	99.5°	...	120	...	38

The opium was stopped, and four ounces of brandy were ordered.

8th.—The respiration was rapid and laboured. Large loud râles were audible all over the chest. He was delirious and restless, and picked at the bed-clothes. He died at 8 p.m.

The autopsy was made eighteen hours after death by Dr. Fagge. The record is given almost as written by him, and the remarks interpolated are his.

The skin was deeply jaundiced. The lungs were reduced in size by pressure upwards of the diaphragm; both were healthy except that they were soft and œdematous, and that there was some ecchymosis of the back part of the right organ. One gland just below the right bronchus was breaking down into a granular pulp. The condition of the lung was not sufficiently advanced to account for this. The heart was healthy; it con-

tained bile-stained fibrin. The abdomen was little distended. On opening it, the first thing seen was the liver, which projected across the epigastrium; it showed numerous clusters of suppurating points on its surface. There was general adhesive peritonitis; the intestines were matted together by lymph, which in parts was slightly opaque or puriform. The omentum was much thickened by infiltration with inflammatory products.

The upper part of the small intestine was moderately distended and showed slight "suction-lines;" the lower part was flaccid. Following the intestine downwards towards the cæcum, one came, at the extreme end of the ileum, upon a circumscribed abscess with thickened walls, lying just below the brim of the pelvis, between the cæcum and the rectum. It was of the size of a walnut, and the appendix opened into it by a truncated extremity. No concretion was discoverable. On cutting through the mesentery thick creamy pus flowed out of the mouths of all the smaller rootlets of the portal vein, the coats of which were also thickened and of a slaty colour.

On dissecting out the trunk of the portal vein it was found as thick as one's little finger, and distended with soft puriform thrombus. This condition extended into the liver itself, the branches of the vein on any section being found full of pus. On the other hand, it did not penetrate into the splenic vein, the mouth of which was closed by a firm yellow piece of thrombus.

All parts of the liver were full of patches of suppuration, but there was no abscess as large as a pea, so far as was observed. The hepatic tissue in the suppurating parts was brownish, in other portions bright yellow.

The gall-bladder contained a thin, watery fluid, almost colourless. It became slightly opaque on the addition of nitric acid. This doubtless shows that the pylephlebitis led to some pressure on the bile-duct, so as to prevent the bile from entering the gall-bladder; but it was not sufficient to obstruct the passage of a good deal of bile into the intestine, for the faecal matter there was of a bright yellow colour. I examined the yellow and brown parts of the liver with the microscope, to see if I could make out any difference in the amount of bile-pigment in the cells which might tend to show that the jaundice was due to a disordered functional activity of the inflamed

parts, but I could find none; and it seems to me that the jaundice was the result of the general pressure on the bile-ducts of the whole liver. The microscope showed that some of the portal canals were crowded with leucocytes, even in the parts of the liver that looked healthy. In the neighbourhood of the abscesses all trace of the fibrous tissue in the canals seemed to have disappeared. In a section that I made with a Valentin's knife, angular fragments of hepatic tissue lay embedded in what appeared to be a homogeneous mass of pus, and the liver-cells of these portions were bright yellow in colour, and little altered. It certainly looked as though the pus-cells as they multiplied were eating up the liver tissue. On the other hand, there were some parts of the hepatic tissue which showed extreme granular change in the cells, as though they were undergoing a parenchymatous inflammation and disintegration. The organ weighed 60 oz. The kidneys appeared quite healthy.

*CASE 9.—Gangrenous dysenteric ulceration of the colon ;
multiple abscesses of the liver.*

C. M—, æt. 24, a waterman, was admitted under my care into the Seamen's Hospital, on October 22nd, 1880. He was an Englishman, and had never left this country.

Previous history.—He was a hard drinker, but his health had always been good.

Present illness.—Commenced about three weeks before admission. He then noticed that he was passing blood and slime with his fæces. The bowels at first did not act more frequently than natural, but on the eighth day before he came into hospital they were moved eight or nine times during twenty-four hours. This frequent action had continued since. He had not been drinking lately. He had been under medical treatment for the last three or four days.

Condition on admission.—The patient looked as though suffering from fever. He was markedly drowsy and apathetic. He lay in bed on his back, taking no notice of his surroundings, but could easily be roused, and then answered questions to the point; soon, however, he relapsed into apathy. His pupils were contracted. There was no jaundice. His tongue was

dry, furred in the centre, with red edges. The appearance of the abdomen was natural. There were no typhoid spots. Liver dulness commenced between the fifth and sixth ribs and extended two fingers' breadths below the costal margin. There was tenderness over the liver below the ribs, and also over the right side of the abdomen, but it required somewhat deep palpation to bring it out.

The spleen could not be felt, and its dulness was not increased.

The cardiac sounds were feeble but normal.

The pulse quick and weak.

The lungs normal.

The urine clear; reaction acid; containing neither albumen nor sugar. Temp. 98.4°.

He was ordered—

℞ Pulv. Cretæ Aromat., gr. xv,
Pulv. Cretæ Aromat. cum Opio, gr. xv,
Sp. Ammon. Aromat., ℥xij,
Aquam ad ʒj. 4tis horis.

Progress of the case.—October 25th.—He still lay in the same drowsy, apathetic condition; indeed, he remained so during the whole of his illness. The bowels had acted six times during the previous night, and four times to-day. The fæces were loose, "pea-soupy," but contained neither blood nor mucus. They were in fact typhoid in character. There was a good deal of tenderness over the right side of the abdomen, and ill-defined increased resistance below the right ribs in front. He was ordered a starch enema with 30 minims of tincture of opium to check the diarrhœa.

				Temp.
M.	.	.	.	101.2°
E.	.	.	.	102.4°

26th.—Condition much the same. One action of the bowels.

				Temp.		Pulse.
M.	.	.	.	99.6°	...	108
E.	.	.	.	103°		

27th.—There had been three actions of the bowels, of the same character as before.

	Temp.	Pulse.
M. . . .	100·8°	... 108
E. . . .	101·4°	

28th.—Four actions of bowels. Fæces frothy, yeasty, and offensive. No spots.

	Temp.	Pulse.
M. . . .	101·4°	... 116
E. . . .	101·6°	

29th.—Bowels had acted three times. No change in character of evacuations.

	Temp.
M. . . .	101·4°
E. . . .	102·4°

30th.—There was some swelling on the right side below the costal margin in the mammary line. There was dulness and tenderness over this area, with considerable enlargement of the superficial veins.

	Temp.	Pulse.
M. . . .	100·4°	... 120
E. . . .	100·2°	

31st.—Hæmorrhage *per anum*. Blood considerable in amount, and bright red. This was the case with each of the three actions of the bowels which had taken place since the last note.

Ordered—

℞ Acidi Gallici, gr. xx,
Glycerini, ʒj,
Aquam ad ʒj. 4tis horis.

As this failed to check the bleeding, a turpentine enema was administered. Brandy ʒvj per diem.

	Temp.	Pulse.
M. . . .	98°	... 128
E. . . .	101·2°	

N.B.—Hæmorrhage had occurred before the morning temperature was taken.

November 1st.—The bleeding ceased after the injection.

	Temp.	Pulse.
M. . . .	100·8°	... 128
E. . . .	102·8°	

2nd.—The swelling below the right ribs had now obviously increased, and obscure fluctuation was detected. The abnormal dulness extended as low as the level of the umbilicus in the mammary line. The line of dulness was not uniform but appeared to correspond to a rounded swelling projecting downwards from the liver. Colon resonance could be detected posteriorly. Exploration was made with a small aspirator needle, and a few drops of thick curdy pus were obtained. The bowels had acted twice but there had been no blood.

	Temp.	Pulse.
M. . . .	100·8°	120
E. . . .	102·8°	

3rd.—The patient being placed under ether, Mr. Johnson Smith opened the abscess by incision, with antiseptic precautions. The liver was found to be adherent to the abdominal wall, and the finger apparently passed into a large, deep, irregular cavity in its right lobe. Only a small quantity of pus mixed with blood was obtained. The wound was dressed antiseptically.

	Temp.	Pulse.
M. . . .	101·8°	120
E. . . .	98°	

In the evening the patient was wandering and restless, and the pulse was feeble. The tongue was dry and furred. There were three loose actions of the bowels.

4th.—The patient passed a quiet night, but did not sleep much. The tongue was still dry and hard. He was still as drowsy and apathetic as ever. The wound was dressed this afternoon; there was but little discharge; the patient then said he felt more easy. The bowels acted once; there was no blood in the evacuation, but some material that looked like pus.

	Temp.	Pulse.
M. . . .	97·8°	146
E. . . .	99°	

5th.—Passed a better night. No pain. Tongue was not quite so dry and hard. The drowsiness continued. The wound was dressed at 1 p.m., and washed out with a solution of carbolic acid. There was little or no discharge.

Ordered Quiniae Sulph., gr. ij, t.d.s.

The subsequent progress of the case presented little change. The patient continued in much the same condition from day to day, except that he gradually became more and more apathetic. The edges of the wound became sloughy and unhealthy, and a feculent smell attended the slight discharge that took place. No pus was discovered in the evacuations. The hepatic dulness did not diminish. He died on November 17th without any noticeable change having taken place.

The following was the range of temperature and pulse :

			Temp.		Pulse.
November	5.—M.	. . .	98·6°	...	138
"	E.	. . .	101·4°		
"	6.—M.	. . .	99·2°	...	132
"	E.	. . .	103°	...	152
"	7.—M.	. . .	100·2°	...	142
"	E.	. . .	102°		
"	8.—M.	. . .	99·4°	...	138
"	E.	. . .	102°		
"	9.—M.	. . .	99·8°	...	138
"	E.	. . .	100·2°		
"	10.—M.	. . .	99·2°	...	132
"	E.	. . .	99·6°		
"	11.—M.	. . .	99°		
"	E.	. . .	99·8°		
"	12.—M.	. . .	99·2°		
"	E.	. . .	102·4°		
"	13.—M.	. . .	101·2°		
"	E.	. . .	101·2°		
"	14.—M.	. . .	98·8°		
"	E.	. . .	100·4°		
"	15.—M.	. . .	97·6°		
"	E.	. . .	100°		
"	16.—M.	. . .	100°		
"	E.	. . .	97·4°		
"	17.—M.	. . .	97·4°		

The autopsy was made by myself.

The body was fairly nourished.

The heart healthy.

The lungs, healthy, except that there was a good deal of collapse of both lower lobes.

On opening the abdomen, the colon as far as the splenic flexure was adherent to the abdominal wall, so as completely to shut off the upper part of that cavity from the lower. There

was no peritonitis, either old or recent, below the adhesions. The ascending and transverse portions of the colon were so soft, that in spite of great care in separating the adhesions, the cavity of the bowel was torn into at numerous points. No communication could be made out between the colon and the cavity of the abscess. The coats of the large bowel were nearly half an inch in thickness at the cæcum, gradually thinning off in passing downwards.

The whole of the large intestine, including the rectum, presented an advanced condition of dysenteric ulceration. The disease was most extreme at the cæcum, though from this point to the upper part of the descending colon the mucous membrane either had been or was in process of sloughing. In the cæcum, ascending and transverse colon, it was gangrenous, pultaceous, and foul smelling, separating in large patches, some of which were loose in the cavity of the bowel. The condition gradually became less intense and it passed almost abruptly at about the centre of the descending colon into a very early stage of the disease. Here it could be distinctly made out to commence in the solitary follicles, many of them being softened in the centre into minute shallow ulcers. Here, also, larger shallow ulcers of the size of split peas were found, and in parts they were more closely aggregated together into crescentic forms, or irregular patches. Within an inch of the anus were two large ulcers.

The stomach and small intestines were healthy throughout.

The spleen presented a few small wedge-shaped patches abutting on the surface.

The liver was not enlarged, but was studded throughout with abscesses varying in size from a pea to a hazel nut; these were most numerous in the right lobe but there were many in the left. One larger than the rest, but not exceeding a hen's egg in size, communicated with the incision in the abdominal parietes. The abscesses were met with in all stages of formation; at some points there was merely a blackish discoloured patch surrounded by a halo of congestion; at others there were distinct sloughs. The main trunks of the portal vein were healthy and free from clot. There was no plugging of either of the mesenteric arteries.

The lesions of the liver, which were rather patches of local

gangrene than abscesses, appeared to correspond with the like condition of the colon.

CASE 10.—*Abscess in front of the bladder, burrowing upwards and opening into the colon, and thence into the left loin (?) ; chronic suppurative peritonitis ; empyema ; pylephlebitis (partially cured) and thrombosis of the portal vein ; infarctions of the lung ; lardaceous spleen and intestine.*

(Reported by Mr. T. CARDWELL.)

W. H—, æt. 18, assistant to a brush-maker, was admitted on September 17th, 1880, under the care of Mr. Howse.

Previous history.—No known illness nor injury before his present disease.

Present illness, dating from his admission, commenced, seventeen weeks before, with pain in the left groin. Hard lumps then appeared in the groin and left loin. These were poulticed, and the swelling in the side burst in two days, and in the course of another five days that in the groin followed. Five weeks from the beginning he suffered from shooting pain in the left side. He had been jaundiced three or four times during his illness, the last occasion being eight weeks prior to coming in.

Condition on admission.—The patient was thin, pale, and anæmic, his face wasted, with yellowish pallid complexion. The conjunctivæ were pearly. In the left loin, about an inch and a half above the centre of the iliac crest, was a swelling, measuring about 4 inches by $2\frac{1}{2}$, with an opening discharging very foul greenish pus. The skin was thin and purple for some distance around. About midway between the preceding swelling and the left anterior superior spine was another, of about the same size, in which fluctuation could be made out, and which was œdematous. There was also another opening in the left groin discharging greenish pus, the surrounding skin being also adherent and purple.

The tongue was moist and furred. The urine was normal.

Progress of the case.—September 18th.—Under chloroform, with antiseptic precautions, Mr. Howse made an incision two

inches long into the upper and posterior swelling, and a smaller opening into the anterior. He passed his finger into the wound, and found that the two communicated. He discovered that the cavity was lined with caseating granulation material, which was then turned out. The finger passed downwards and backwards to muscles, but no exposure of the vertebræ was detected. A probe was also passed forwards in the direction of the inguinal opening. A drainage tube was inserted.

The subsequent history of the case as detailed is very much that of the progress of the abscess, which exhibited many variations. On October 10th it is noted that there was no discharge. On October 20th fluctuation was detected just above the iliac crest. On October 24th a large quantity of pus was evacuated from this situation by incision. On November 24th the patient was "going on well." On December 29th there was fulness and swelling in the right groin. On January 15th this was opened and a large quantity of sanguineous pus let out. On February 20th another abscess appeared near the left iliac crest. On February 24th another abscess was opened on the left side just below the ribs, and fæcal matter appeared in the discharge. With a slight interval of improvement during the beginning of March, the patient gradually sank and died on the 12th of that month.

It is obvious that any symptoms due to the hepatic affection which was discovered on post-mortem examination, were completely overshadowed and masked by those due to the huge burrowing abscess. It would be tedious to relate them, and of absolutely no use from the point of view of this paper to detail them. They consisted mainly of pain of varying intensity, chiefly in the left side, occasional sweating, and a febrile temperature throughout, which fluctuated between 98° and 104°, being usually about 100°. Towards the end the symptoms of peritonitis are recorded. The urine was very repeatedly examined, and only on one occasion contained albumen, which was then in large quantity. No record of the passage of pus *per anum* is given. The treatment adopted was mainly that of subcutaneous injections of morphia, of good nourishing diet, and a moderate amount of stimulants.

I have thought, however, that the case is well worthy of being placed on record amongst this series, both as indicating

the possible curability of pylephlebitis, and also the occasional obscurity in the diagnosis of caecal disease.

The autopsy was made twenty-two and a half hours after death by Dr. Goodhart. I have copied the record almost exactly as written by him.

The body was much emaciated; there was slight œdema about the ankles.

There was a sinus in the right groin, which opened into an abscess behind the pubes; two others in the left groin, which had closed all but the superficial sore; and another in the left loin, which opened into the descending colon.

The right pleura contained about half a pint of pus; the surface was coated with yellow lymph, and its lymphatics were full of a milky fluid.

The greater part of the lower lobe of the right lung was solid, and the lymphatics over it injected. At one spot in the lower edge, at the hinder part, the lung tissue was softening. There were also several small infarctions at the same edge, the lung tissue being brick-red and separated from the rest of the organ by a line of demarcation of a pale colour. In the lower part of the same lobe the lymphatics were distended, in one case with pus, in others with inflammatory formation, either in the vessels themselves or following their course. In connection with this the lymphatic glands on the right side of the mediastinum and in the fork of the trachea were all swollen, some of them red and fleshy from an early inflammation, others paler, but still fleshy, from a more chronic disease.

The left ventricle was somewhat thickened, but the valves were healthy; there was no *ante-mortem* clotting.

The vena azygos was unusually large and distended.

The diaphragm was adherent both to the base of the lung and the liver.

The peritoneum presented adhesions all over of a somewhat chronic kind, those between the liver and diaphragm being especially tough. Pus was imprisoned in the loculi of the adhesions.

In the lower part of the abdomen behind the pubes, between it and the intestinal coils, was an abscess of old date, with thickened, discolored walls. It ran down to the right and surrounded the appendix cæci, and also to the left by the side of

the colon. The surrounding tissues and the mesentery were thickened and discolored. The abscess communicated with the sinus in the right groin, and had also extended into the recto-vesical pouch, and opened into the rectum about one inch and a half from the anus.

The interior of the lesser bowel was lardaceous in places. No ulceration was found in it, until about one inch from the ileo-cæcal valve, when two small ulcers or, rather, holes in the mucous membrane, were found. These communicated with thickenings externally, and their appearance was in favour of their being produced by burrowing from without inwards.

The condition of the cæcal appendix was interesting. It lay surrounded by a part of the abscess of oldest date. Near its tip was an oval depression, into which a probe passed for about two millimètres, not, however, quite into its canal. On opening it up, the mucous membrane was puckered and discolored over it, but not perforated. "I am inclined, however, to think, from the appearance of the parts, that there may have been formerly some ulcer of the mucous surface now healed, and that this may have been the origin of all the mischief."

The colon was healthy as far as the splenic flexure, but just below this was a sinus leading straight into the loin, and still further down several puckered patches, looking like the scars of old ulcers, with some actual ulcers still remaining. They led into the thickened and discolored mesentery, and were in close proximity to the extension upwards of the pelvic abscess. Here, again, it was difficult to decide whether they were primary in the bowel, or produced by extension from without.

The liver weighed 59 oz. When its surface was cleared of lymph, pus, and adhesions, several depressions were seen. These, on section, proved to be scars, several of which had small yellow central deposits, the relics (?) of abscesses of some date. One abscess was half an inch in diameter, altogether in the caseous state, without any scar tissue. In addition to these there were numerous points throughout the liver of grey cirrhotic-looking material, with two or three central spots of yellow, which looked like some early caseous degeneration of fibroid matter. But it was at once noticeable that all these were in the portal canals; pus exuded from some.

The portal vein, in the fissure, was completely blocked, its walls much thickened, and its canal full of soft clot, with some yellow, bile-coloured pus in the centre. The yellow points in the liver were therefore the result of diffused pylephlebitis of some standing, which had to some extent recovered.

With regard to the collateral circulation, there was no *caput medusæ*, and nothing remarkable about the stomach or intestines. The œsophagus was free from dilatation of its veins. There was, however, a plexus of large veins in the portal fissure, in front of the portal vein, which probably communicated with branches from the vena cava, and so relieved the circulation by a direct passage. The peritoneum was in such a condition from adhesions that it was impossible to make sure of the whole collateral circulation, but there was no undue enlargement of the left renal vein, as has been noted in other cases. The plexus of veins in the portal fissure was examined afterwards. Some of them ran forwards in the portal fissure; others ran backwards, and were lost in the tissues surrounding the diaphragmatic opening of the vena cava.

The spleen weighed 11 oz. It had suffered in the same way as the liver, in that its vein was plugged and full of pus. There was an abscess in its tissue arising from this cause. It was also lardaceous.

"I am inclined to view the case as one of ulceration of the cæcal appendix, giving rise to obstruction in the portal veins, to pylephlebitis, and this to ulceration of the colon."

A microscopic examination of the liver was made by Dr. Goodhart, and I subjoin an extract taken from the 'Pathological Transactions' for 1881. He says:

"Under the microscope a quantity of new fibrous tissue was seen to have destroyed the liver substance almost completely at the spots described as scars."

"The interest of the case seems to hinge on the probability that suppurative pylephlebitis had ended, not as usual in the death of the patient, but in cicatrization, and in doing so had set up isolated centres of cirrhosis, such as have been described by Hanot, Charcot, and others in connection with the ducts. A condition similar to this has been recorded by Dr. Moxon and myself, to exist occasionally in the kidney."

CASE 11.—*Gall-stones; inflammation and contraction of the gall-bladder and surrounding parts; dilatation of the hepatic ducts; suppurative inflammation of the left lobe of the liver.*

(Reported by Mr. HENRY DAVY.)

M. T—, cook, æt. 45, was admitted into Bright Ward on December 29th, 1876, under the care of Dr. Moxon.

Family history.—Unimportant.

Previous history.—She enjoyed good health up to seven years previously. She then began to suffer from epigastric pain. This she described as causing her “perfect agony,” and as coming on every evening, lasting for twelve hours, and leaving her weak and exhausted. At the same time she also suffered from vomiting after food.

Five weeks after the commencement of the pain complete jaundice supervened. She became an in-patient of University College Hospital, whence she was discharged at the end of thirteen weeks quite cured. During her stay in the hospital she vomited on several occasions about half a pint of blood, and she also passed *per anum* two hard masses, one of the size of a “caper,” the other larger and of a square shape with pointed edges.

She had never been in the habit of drinking, nor had she ever suffered from morning sickness, on the contrary, breakfast was always her best meal.

Present illness.—The past twelve months she had been ailing more or less, being unable to do her work and vomiting after eating meat. Three weeks ago, whilst serving dinner, she fainted, and was afterwards very sick; the sickness had increased since, and she was unable to retain solid food. The vomited matters were yellow, and of a very bitter taste. She suffered great epigastric pain, especially towards the afternoon.

Condition on admission.—The heart and lungs were normal. The conjunctivæ were jaundiced. The abdominal muscles were particularly tense. The hepatic dulness commenced in the fifth intercostal space, and reached to one inch below the margin of the ribs. The urine had a sp. gr. of 1020, was high-coloured, showed to tests a trace of bile, and contained neither albumen nor sugar. Pain of a throbbing character, and continuous for some hours after its commencement, was

felt across the lower part of the chest in front, extending from a point two inches below the nipple and reaching right across. There was also tenderness to percussion over the spines of the middle dorsal vertebræ.

Progress of the case.—December 30th.—Morning temperature $101\cdot8^{\circ}$, pulse 102.

31st.—Morning temperature $102\cdot4^{\circ}$, pulse 100.

January 1st.—She did not sleep well, and suffered much nocturnal pain. She often vomited. There had been no action of the bowels since December 29th. Morning temp. 100° , pulse 100.

2nd.—No change. She took solid food for dinner.

				Temp.		Pulse.
M.	.	.	.	$100\cdot6^{\circ}$...	100
E.	.	.	.	$102\cdot7^{\circ}$...	102

3rd.—Meat for dinner; no sickness. Did not sleep well.

				Temp.		Pulse.
M.	.	.	.	$99\cdot2^{\circ}$...	96
E.	.	.	.	$100\cdot8^{\circ}$...	100

4th.—Although taking solid food, she had not vomited the last few days. She slept better last night. She was now free from pain, but felt very weak. The liver was still¹ rather tender on percussion. The urine was of sp. gr. 1014; it showed distinct traces of bile, but no albumen nor sugar.

				Temp.		Pulse.
M.	.	.	.	$99\cdot4^{\circ}$...	94
E.	.	.	.	$100\cdot4^{\circ}$...	96

5th.—She relished her food, and had not been sick. After an aperient pill the bowels acted six or seven times last night. She had a hot and cold flush last night.

				Temp.		Pulse.
M.	.	.	.	$100\cdot4^{\circ}$...	106
E.	.	.	.	$101\cdot8^{\circ}$...	100

6th.—She complained of “disagreeable taste in her mouth,” and was very thirsty. She vomited once in the night, and the

¹ Although the word “still” is here used, there is no previous note of this symptom in the record.

ejected matters were very bitter. She woke up once sweating, and this was followed by hot and cold flushes.

				Temp.		Pulse.
M.	.	.	.	101.2°	...	96
E.	.	.	.	101.8°	...	90

7th.—

M.	.	.	.	99°	...	84
E.	.	.	.	99.4°	...	78

8th.—

M.	.	.	.	100°	...	—
E.	.	.	.	100.2°	...	—

9th.—At 8 p.m. last evening she was seized with great pain in the right side, the continuance of which kept her awake all night. In the morning the hepatic region was very tender to percussion, and the liver could be felt of stony hardness two inches below the ribs. The urine showed traces of bile, but contained neither albumen nor sugar.

				Temp.		Pulse.
M.	.	.	.	101.8°	...	102
E.	.	.	.	101.6°	...	104

10th.—She had been sick three times since 6 a.m.; this relieved her pain; the vomited matter was of a dirty, dark green colour. Tested by concentrated nitric acid and Pettenkofer's test it showed abundance of bile; the microscope revealed nothing but amorphous granular matter. The urine had a sp. gr. of 1015, and contained much bile.

				Temp.		Pulse.
M.	.	.	.	101.1°	...	112
E.	.	.	.	103.6°	...	102

11th.—

M.	.	.	.	101°	...	102
E.	.	.	.	101°	...	104

12th.—The patient continued to vomit from time to time, chiefly in the morning. Usually she was attacked with great pain, and this being followed by sickness she was relieved. She had twice vomited this morning. She slept better last night, and her pain was now less. The vomit was very watery, of a yellowish-brown green colour; it reacted for bile; the microscope only revealed bile-stained epithelial *débris*.

	Temp.	Pulse.
M. . . .	95.6°	84
12.15 P.M. . .	97.2°	—
E. . . .	98.6°	96

13th.—Last night she had a severe rigor, and slept very little. She vomited this morning, and the ejecta contained but little bile. The urine contained a good deal of bile, and an excess of lithates, but neither albumen nor sugar.

At 12.15 p.m. she had a severe rigor, during which she was perfectly conscious, and said she was not in any pain but felt weak. Her axillary temperature at that time was 95.8°.

At 1.10 p.m. she was still conscious and complaining of weakness. She said she was in want of sleep, and asked for a morphia injection.

At 1.20 p.m. the temperature in the rectum was 111°. She was pulseless and gasping for breath; the gasping increased in intensity most rapidly, and at 1.23 she died, the heart stopping at this time.

The autopsy was made two hours after death by Dr. Fagge. The body was fairly nourished. There was no jaundice. The heart and lungs were healthy, except that the latter were slightly emphysematous.

On opening the abdomen the stomach was found adherent to the under surface of the liver, by what proved to be recent adhesions. The duodenum was puckered up and fixed to the gall-bladder and adjacent parts by old fibrous tissue. The left lobe of the liver was obviously enlarged and projected downwards; it was soft and fluctuating.

After removal of the parts Dr. Fagge laid open the stomach and duodenum and found them healthy. The orifice of the common bile-duct was rather unusually prominent. A probe, on being passed into it, emerged upon the mucous surface of the intestine again, about one third of an inch higher up. This second orifice was smooth and old, and about the diameter of a pea; but it would doubtless have stretched to much larger dimensions. Evidently it had been caused by a gall-stone ulcerating through. The probe could easily be made to pass up into the common bile-duct, and this being done the duct was then dissected out. It was found to be enormously thickened and dilated, at least the size of a fore-finger, and the walls were

as crisp and thick as those of a large artery. It was completely embedded in adhesions, and the gall-bladder was placed at right angles to it. This latter was contracted and reduced to a mere relic, its cavity was divided into three or four pouches communicating with one another, and containing a colourless mucus. There was no calculus either in the gall-bladder or in the common bile-duct. The latter contained yellow bile. The tissue of the liver was healthy. The bile-ducts in it were everywhere slightly enlarged; those in the right lobe were perhaps half the diameter of the branch of the portal vein, by the side of which they ran; in the left, they were much more considerably dilated, exceeding in size the corresponding portal radicles.

On incising the prominent left lobe of the liver, its lower half was found to be converted into a mass of small abscesses, communicating freely with one another and containing a greenish pus. There was also some sloughing of the tissue. Dr. Fagge adds, "I could not definitely make out the relations of the abscesses to the bile-ducts, but from the way in which their channels appeared to branch, I came to the conclusion that they had probably arisen in dilated branches of the ducts, enlarged out of proportion to those in the rest of the liver. This I think I have before seen to be the case when there has been obstruction to the out-flow of bile."

The kidneys were healthy.

CASE 12.—*Gall-stones; old obstruction of and inflammation around the gall-bladder; fibroid phthisis; suppuration of ducts within the liver; pleuritic effusion.*

(Reported by Mr. J. C. UHTHOFF.)

J. H—, æt. 52, married woman, occupation, household duties, was admitted, on November 23rd, 1877, under the care of Dr. Wilks; subsequently she came under Dr. Pavy's charge on his taking over the clinical wards.

Family history.—Her mother died of cancer of the breast.

Previous history.—She had been married twenty-six years, and had had four children and two miscarriages. She contracted typhus fever when twenty-seven years old. Seven years

ago she had a severe attack of variola, and she said she had never been well since this.

Present illness.—Six weeks ago was suddenly seized with choking whilst talking with her friends; they struck her on the back and she recovered. Her doctor said they probably dislodged a gall-stone. Five weeks ago she became ill with symptoms of a severe “feverish cold,” pains in her limbs, &c. She took to her bed, and had not been up since. She had gradually become worse up to her admission, but her friends could not specify any marked change as having taken place at any particular time. The general character of her symptoms had been as follows:—Jaundice throughout; from time to time severe paroxysmal epigastric pain; progressive wasting. There had been no sickness. There had been only two actions of the bowels during the past five weeks, and these only after medicine and enemata; the motions were clay-coloured. She had been passing high-coloured urine. She had lain in bed, taking but little notice of what had been going on around her, and every night she had been delirious. Four weeks ago she commenced to lose her power of hearing, and she was now very deaf.

Condition on admission.—She was wasted and cachectic. She lay low down in the bed, with her knees drawn up as though suffering from abdominal pain. Her eyes were closed, and she was extremely apathetic; even the clinical examination scarcely attracted her attention. She was constantly uttering low, short groans, and at times placed her hand on her abdomen as though in pain. Her skin was very anæmic, with a slight jaundiced tinge; it was warm, and in parts moist. Her lips and tongue were dry, and her teeth were encrusted with sordes. Her breath was fetid; she was very thirsty. There was complete anorexia, but no sickness.

Her abdomen was somewhat distended, but no fluid was detectable; it was tender, but not to a marked degree, and not at all in the hepatic region, nor was there any muscular rigidity. Hepatic dulness extended from the third rib to one finger's breadth below the costal margin. The splenic dulness was normal in extent. Her pulmonary physical signs were normal over the front of the chest, but she was too ill to examine behind. Respirations 30.

The heart's apex beat in the fourth intercostal space. The

cardiac sounds were indistinct. Her pulse was moderately full and very compressible, the number 108.

The urine had a sp. gr. of 1017, and was loaded with lithates. There was a play of colours with strong nitric acid, but no reaction with cane sugar and concentrated sulphuric acid. It contained neither albumen nor sugar. The temperature was 97.4° .

Progress of the case.—November 24th.—The bowels acted after an enema; the fæces were clay-coloured. Temp. 99.2° .

It would be tedious to insert here the notes of the case from day to day. No very noticeable change took place, and one day's account reads very much like another's. She steadily went on to a fatal termination, with one or two intervals of apparent improvement. I will indicate the range of temperature at the end of the record, and will note down the facts that seem to be of most importance.

28th.—She was apparently neither better nor worse. During the day she was quiet and appeared to sleep at intervals, but towards evening she became delirious. Her mouth and tongue were both very dry. The abdomen was neither hard nor tender. The jaundice continued. The pulse was soft and compressible.

29th.—She expressed herself as feeling better, and she really seemed so. She was quieter last night and not delirious. The skin was warm and natural, but still jaundiced. Her tongue and lips were more moist. The pulse was stronger, its number 120.

30th.—She was restless and delirious again last night. Her tongue was extremely dry, and thirst was excessive. There was still slight tenderness on deep palpation of the abdomen.

December 3rd.—There was more tenderness in the right hypochondrium than in any other region of the abdomen.

4th.—The tongue was dry and brown. The sordes were again collecting about the teeth. There appeared to be a very slight bulging of the side of the chest in the hepatic region, but measurement showed a difference of barely half an inch.

5th.—She was lying "in a heap" in bed, on her back. Her face was flushed. Her tongue was dry and brown. She had sordes on the teeth. She was extremely apathetic, and did not as usual ask if she was better. She was very anæmic. There was no jaundice. Altogether she seemed much worse.

8th.—She was seldom delirious at this time. She appeared to be gradually sinking, and became more emaciated every day.

11th.—She seemed somewhat improved. She was less anæmic and rather brighter. The sordes collected in less degree about her teeth. Her abdomen was still full, but there was no fluctuation. There was tenderness over the liver. The hepatic dulness extends two fingers' breadths below the ribs.

13th.—She was very low.

15th.—There was a marked improvement in the general condition. She was brighter, less deaf, and much less cachectic in appearance.

17th.—She seemed to be gradually picking up strength.

22nd.—Was much the same. Did not complain of pain. The tongue was moist but corrugated. The abdomen was tender only in the hepatic region. There seemed to be a good deal of hardness below the right ribs. There was no further increase of dulness.

27th.—She looked more cachectic, and sank low in the bed. She was again somewhat jaundiced.

January 1st.—The patient seemed very much worse. She was drowsy and had a troublesome cough.

2nd.—In the early morning she had a good deal of pain in the upper part of the right side of the abdomen. She sank low in the bed with her knees drawn up.

4th.—The patient was in a drowsy state. Urine sp. gr. 1010; decided play of colours with nitric acid.

5th.—She suffered from severe pain in the right side of the abdomen at the upper part, and was calling out with it.

6th.—Her face was pale and slightly jaundiced. The urine reacted for bile with nitric acid, and also with Pettenkofer's test. The surface was clammy, and she appeared to be sinking. She died at 5.30 p.m.

Date.		Temp.	Pulse.	Resp.
November 23—M.	. .	97·4°	... 108	... 30
„ 24—M.	. .	99·2°	... —	... —
„ 26—M.	. .	97·6°	... —	... 32
„ 27—M.	. .	98·6°	... 100	... 35
„ 28—M.	. .	—	... 116	... 28
„ 30—M.	. .	100°	... 112	... —
December 1—M.	. .	99·2°	... 120	... —
„ 3—M.	. .	97·6°	... 100	... 28

Date.		Temp.	Pulse.	Resp.
December	3—2 P.M. . .	102°	...	—
	E. . .	101.2°	116	—
"	4—M. . .	98.8°	106	25
	E. . .	99°	100	—
"	5—M. . .	103.6°	130	28
	E. . .	98°	100	24
"	6—M. . .	98°	112	25
	E. . .	99.2°	112	28
"	7—M. . .	99.2°	112	—
	E. . .	100.2°	120	—
"	8—M. . .	100°	115	25
	E. . .	100.2°	120	28
"	9—M. . .	100°	—	—
	E. . .	100°	120	28
"	10—M. . .	98.2°	108	30
	E. . .	98.6°	128	30
"	11—M. . .	98.4°	110	26
"	12—M. . .	100.2°	120	28
	E. . .	97.8°	134	—
"	13—M. . .	98.4°	106	28
"	14—M. . .	98.7°	—	—
	E. . .	98.4°	—	—
"	15—M. . .	98.4°	112	—
"	17—M. . .	99.7°	110	—
"	18—M. . .	98.6°	100	—
"	19—M. . .	100.4°	106	—
"	20—M. . .	99.2°	106	—
"	22—M. . .	100.4°	110	—
"	24—M. . .	98.4°	105	—
"	26—M. . .	98.2°	—	—
"	27—M. . .	100.5°	110	—
"	28—M. . .	99.8°	120	—
"	29—M. . .	98.4°	108	—
"	31—M. . .	99°	112	—
January	3—M. . .	101.5°	131	—
	E. . .	103°	130	—
"	4—M. . .	103.2°	130	—
	Afternoon . .	102.8°	—	—
"	5—M. . .	102.8°	136	—
"	E. . .	100.2°	128	30

Autopsy, twenty-one hours after death by Dr. Goodhart. The remarks at the end of the report are by him.

The body was emaciated. There was a slightly yellowish tinge of skin, without decided jaundice.

The brain was healthy.

The right pleura was full of serous fluid. The surface was covered with a somewhat thick layer of lymph. The left was healthy.

The right lung was solid throughout, and very small, as if from compression. On section it showed that, in addition to changes due to pressure, there was extensive thickening of the interlobular septa throughout the lung, as well as some pneumonia near the centre. There was no doubt that a chronic inflammation of the whole organ was in process, and would have led to a fibroid lung. The base was firmly adherent to the diaphragm, and the latter to the liver. It seemed probable that the pulmonary was secondary to the hepatic affection.

The heart weighed 8 oz. and was healthy.

The femoral and pelvic veins were normal.

On opening the abdomen, the liver was found to be a little enlarged downwards, and toughly adherent to the abdominal wall in the neighbourhood of the gall-bladder. On its removal it was found to be firmly adherent to the diaphragm over the whole of the right lobe, and the under surface was also firmly adherent to the kidney and hepatic flexure of the colon, so that they were not easily separated. From this it was evident that there had been a very extensive old inflammation all around these parts. It appeared that the chronic thickenings above detailed centred round the gall-bladder, which was shrivelled to a relic, with hardly a trace of cavity. The cystic duct was dilated, as were all the ducts throughout the liver, and the latter in the smaller branches were suppurating extensively. Thus the liver on section had the aspect of being studded with small abscesses, and also with cavities containing inspissated bile and gritty matter. There was a gall-stone, of round shape and a quarter of an inch in diameter, situated in a sort of pouch at the side of the common bile-duct beyond the portal fissure and behind the duodenum; it did not obstruct the duct at all. A probe passed along the duct before opening it hardly gave notice of its presence. The orifice of the duct in the duodenum was smaller than usual, though quite free. The weight of the liver was 72 oz. The portal veins was healthy. The stomach was healthy. The spleen weighed 10 oz. and was healthy. The adrenals were healthy.

“With regard to the course of events in this case, I must

suppose that there had been originally some considerable obstruction by gall-stone, sufficient to lead to dilatation of the ducts and suppuration, and that afterwards the obstruction had disappeared, so that the jaundice towards the end and the death itself were caused by the multiple hepatic abscesses."

CASE 13.—Carcinoma of the common bile-duct, obstructing its entrance into the duodenum ; suppuration of the hepatic ducts within the liver ; no secondary growths.

My personal knowledge of the present case dates only from the post-mortem examination, and I am indebted for such particulars of the history as it was possible to obtain to Mr. Fortescue Ingram, late one of the medical officers of the Chelsea Union Infirmary, under whose care she came in that institution. The patient was a dressmaker, æt. 41, and was admitted on November 12th, 1879.

Previous history.—She had never suffered from any symptoms of biliary colic. She had not been addicted to drinking.

Present illness.—She stated that it commenced with what she called a "severe bilious attack," three months prior to her admission. This she had neglected, and she had become gradually worse up to her coming into the infirmary.

Condition on admission.—She was much emaciated, and complained of great prostration and weakness. Her skin was deeply jaundiced, of an olive green colour. The faces were clay-coloured. Every other point was negative. There was no pain nor enlargement of the liver, nor tenderness over the organ, nor ascites. There was no vomiting, and the bowels acted regularly.

Progress of the case.—During her stay in the hospital she was always cheerful, and expressed herself from day to day as feeling better. She was never delirious. Her temperature was never febrile, but it was not noted regularly. Her emaciation, jaundice, and weakness progressively increased however, and for the last few weeks of her life she suffered from copious, offensive perspirations. No new symptom presented itself, and she gradually died out on February 7th, 1880, after an illness altogether of six months. On the day of her death the following note was made:—"Sinking rapidly. Lying in a calm, unconscious state, simulating sleep. No delirium."

The autopsy was made twenty-four hours after death, by Mr Ingram and myself.

Body much emaciated and very deeply jaundiced ; no enlarged glands. On opening the abdomen the parts in the right hypochondrium and epigastrium were found matted together by old adhesions, the transverse colon fixed to the liver, and a moderate collection of purulent fluid between these two viscera. The gall-bladder was greatly distended, holding perhaps half a pint or even more. On incising it a clear, yellowish, serous fluid escaped ; this was not purulent.

The liver, stomach, duodenum, and pancreas were removed *en masse*, and the parts carefully dissected out. The hepatic, cystic, and common bile-ducts were greatly distended ; but it was found impossible to pass a small probe through the end of the duct into the duodenum. On opening up the latter viscus, the cause of the obstruction was found to be a hard nodule, one inch in length by half an inch in diameter, occupying the wall of the duct just at its entrance into the duodenum and completely occluding it. This on section was greyish and semi-translucent, hard in some parts, but soft in others, and presenting all the appearance of a cancerous growth of the encephaloid variety.

On the surface of the liver were many dilated ducts, and on section yellowish-green pus poured out from innumerable points, which afterwards were found on dissection to be the dilated endings of the smaller ducts. The portal vein and its branches throughout the liver were found to be quite free from pus and healthy. No gall-stones were discovered. The lumbar glands were somewhat enlarged. On searching for secondary growths, none were found either in the lungs, liver, kidneys, or spleen, or on the surface of the peritoneum. The brain was not examined, but the patient had been throughout free from head symptoms. The head of the pancreas was quite healthy, and in fact the only other morbid appearances were emphysema and œdema of the lungs and a large, soft spleen. Microscopic examination of the growth showed it to be a typical encephaloid carcinoma, consisting of an abundant nucleated stroma, the alveoli of which were crowded with cells.

PES VALGUS ACQUISITUS.
PES PRONATUS ACQUISITUS.
PES CAVUS.

By C. HILTON GOLDING-BIRD, B.A., M.B.

WITH so much that has been written upon orthopædic surgery, and especially that branch of it that deals with deformities of the feet, it may well seem that nothing is left to be added by a general surgeon upon so common a malady as acquired flat foot. Having, however, for some years, kept careful notes of all the cases that have come before me, I find on their perusal, that if there is nothing new to be noted in them, there is yet much which may well bear repetition, both in point of clinical interest and with the ulterior view of treatment.

There are, at the outset, a few general considerations which must not be passed over. Osteologists think very much of the arch of the foot; it is instanced as a beautiful example of natural architecture, and yet when taken alone it is of little, if any, value in sustaining the weight of the body, and this fact the clinical study of the foot clearly substantiates. It is better compared to a strung bow, of which if the string fail it becomes useless either as a spring or to sustain any weight. It cannot be an arch, in the architect's sense of the word, for it has no piers on which its extremities can rest nor by which they can be prevented separating from each other. There is

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The bone changes have been well described by Hueter,¹ but as my cases do not illustrate this part of the subject, I must refer to his description, being content here with just a general statement. As the result of undue weight being thrown upon the foot, the astragalus gradually gets pushed more down on to the anterior process of the calcis, and the sustentaculum becomes less pressed upon; in consequence, as growth proceeds, this last comes to occupy a higher position upon the inner side of the os calcis than normally in the adult, and each year presents a further bar to the restoration of the sinking arch. The astragalus, wedge like, gradually pushes its way forwards and downwards till its head forms a prominence on the inner border of the sole, and then the arch becomes a convexity; while in extreme cases, the os calcis being thrust preternaturally to the outside gets a facette developed upon it from pressure of the external malleolus.

The limit to lateral movement in the adult astragalo-calcanean joint, in the sense of inversion of the foot, is the contact of the sustentaculum tali with the astragalus; and of eversion, the contact of the outer edge of the articular under surface of the astragalus with that of the articular upper surface of the os calcis. Let this last check be decreased by constant overweight, tending to evert the foot, and then the sustentaculum will grow up to a higher level than usual (like its position normally in the infant), and so tends to prevent restitution of position by reinversion of the foot.

Thus is produced, besides a merely flat foot, what is also termed an extrorsed one. A far better expression is pronated foot; for this only refers to the actual position of the foot, whilst extrorsion, in many minds, carries with it an idea of its causation, often falsely considered that of muscular spasm.

The pain of flat foot has especial reference to the distortion, and not only to ligament stretching or to tendon straining. In most cases the pain is referred to below the outer ankle, in a far smaller proportion of cases to the sunken arch pressing on the ground. Without taking such an extreme case, where the os calcis has itself become impressed by the fibula, the localisation of the pain at this spot can be yet explained.

The term "bow" has been used in preference to "arch,"

¹ Hueter. 'Grundriss der Chirurgie,' 1882.

but strictly neither is correct. More correctly the curve is that of a quarter of a hollow sphere, the section through one pole representing the highest point of the curve, whilst the equatorial section rests on the ground. If, then, the polar section gets flattened out whilst the latter is fixed, a compression of the tissues of the equatorial portion must occur; or in the foot, if the arch falls, so as to elongate the inner side of the foot, a "crowding" together of the bones on the outer side will follow; and this seems to explain the pain so constantly being located on the outside. Reasoning thus induced me in four cases, to be related, to remove a wedge from the inner side with a view of relieving the compression to which the outer edge of the foot was subjected. Pain in the same spot is seen in cases of simple extrorsed or pronated foot, from compression of the tissues under the outer ankle.

It will be remembered that the pain of a neglected and unreduced Potts' fracture, when the patient gets about, is below the outer malleolus, and that the foot is extremely everted.

Talipes varus is a supination of the foot and talipes valgus (flat foot) a pronation; but a marked degree of pronation may also exist without there being any flat foot whatever (Cases 19, 27, 37, 43); but where the arch is sunken there must be also pronation.

Although the astragalo-calcanean joint is a very strong one to resist severe or sudden strains, yet it is possessed of marked lateral movement. The lateral movement that is clinically and generally ascribed to the astragalo-tibial joint, really belongs to the astragalo-calcanean, and when unduly present gives rise to the condition of "weak ankles," common enough in girls of poor muscular development, or in that constitutional group from which "lateral curvature" cases arise. The inner malleolus seems in such cases to project (or to "grow in"), whilst the heel of the foot gets thrown in an ungainly fashion to the outer side. This condition is undoubtedly often accompanied by some loss of the arch, but never necessarily so; and if the foot be raised from the ground not only is the arch well formed, but by measurement it can be shown to be normal; whilst the os calcis, if moved from side to side, is felt to have undue mobility at the astragalo-calcanean joint (Case 19).

A number of circumstances unite to produce this; the same

that may bring about lateral curvature of the spine. The ligaments in such constitutions do not possess their usual strength and quickly yield ; there is a weak muscular development whereby the muscles acting on the ankle cannot maintain the equilibrium of the foot, and the weight of the body (which though very likely spare enough) is unduly heavy under existing circumstances.

Attention may well be drawn here to the difference between the *maintenance of equilibrium* and the *support of weight* in the body.

The former is done by the muscular system mainly, the latter by less highly organised structures. The usual example given is that of the ligamentum nuchæ ; which in man, who has his head balanced on his spine, exists only in name ; while it becomes a cord of great size in such animals as the horse or ox. The weight of the head is maintained by it, and the muscles are relieved of unnecessary work.

Standing erect is in the main a muscular act of maintenance of equilibrium ; but " standing at ease " or " halting on one leg " is quite another matter ; in this case the great trochanter on the side on which a man halts, is thrown out against the strong fascia lata that runs from the ilium to the knee, and by its resistance to the bone prevents the body falling sideways. The muscular system now requires only to keep the equilibrium " fore and aft " at the hip-joint ; a matter of no great difficulty, and hence the relief felt by this position.

Whilst then, the support of weight, a continued drag, or a sudden check are the duties of fascial rather than muscular structures, yet the offices of these two are sufficiently correlated that one cannot suffer without the other ; and this is well seen in the case of flat foot.

The strings of the bow are derived, as before stated, from these two structures, and if the ligamentous fail, the muscular, whose office is secondary to that of the ligaments, is quite insufficient of itself to keep up the curve of the foot ; and conversely, where the muscles fail (as in infantile paralysis), the ligaments very quickly yield (Case 12). Where the elastic ligament in the sole yields, the interosseous astragalo-calcanean ligament has undue strain thrown upon it ; it, too, yields ; and the sliding forward of the former bone upon the latter, as before mentioned, commences and a sunken arch results.

Where, however, the ligaments of the sole do not yield, and the arch maintains its shape, and yet the muscles of the leg are, from whatever cause, unable to keep up the equilibrium about the ankle, the foot begins to rock below the astragalus, and the strain upon the interosseous ligament is excessive, and presently the condition of everted or pronated foot is brought about, which has already been referred to.

Omitting cases of valgus before 7 years of age, which are of the true or congenital form or from rickets, the cases of *Valgus Acquisitus* fall under three classes, though transitions from one to the other constantly occur.

1. *Simple acquired flat foot.*
2. *Acquired flat foot with fixation (fixed flat foot).*
3. *Acquired flat foot with arthritic symptoms.*

This is a clinical classification, and really represents stages of one and the same affection, yet it by no means follows that any one form need run into the grade above it; thus, a case in group 1 may be of the severest character and yet never come under group 2.

Under the general remarks that have gone before some account will be found of simple *extrorsed or pronated foot*, without sinking of the arch; and Cases 19, 27, 37, and 43, were referred to as examples of such. The treatment being as for simple flat foot, no further remarks will be made upon it; but a very common provocative of this condition must not be passed over; it is found in the style of boot worn so much by girls; for it is in them rather than in boys that it is seen.

The narrow soled boots, with high heels placed very far forwards, so that the sole of the foot is always on an inclined plane, which in a strong muscular subject are worn with comfort, at once in the more delicate produce "weak ankles."

There is first the narrow sole and heel, on which it is but little easier to balance on a rough road than it is on a pair of skates over a ploughed field; whilst the height that the heel takes the ankle off the ground gives immense leverage from below upon the astragalo-calcanean joint; and this must be met by proportionately powerful muscular development in order to preserve the equilibrium of the foot. When this is not forthcoming the joint gradually yields, and the parents, discovering a projection of the inner malleolus, bring their daughter to be treated for "ingrowing ankle."

The obliquity of the tread or sole of these boots from behind downwards and forwards, strains the interosseous ligament in the antero-posterior direction, and thus tends to let the astragalus forwards and commence the destruction of the arch of the foot.

A flat sole and a low heel, with a bandage round the ankle, will in time suffice in many cases for the surgical part of the treatment, but in more severe cases the use of the spring apparatus, mentioned later on, is requisite.

The *causes* of *Acquired Flat Foot*, apart from the question of boots just mentioned, are the same to whichever of the three classes it belongs; and generally my cases, of which I have careful notes (just fifty in number), show that overstanding or overwalking is the main one. Allied to this is the carrying of heavy weights (Case 9), and in one case valgus was induced in a man, æt. 49, from constantly coming down on one foot in the act of planing (Case 20); and in a lad, æt. 13 (Case 17), the right foot suffered from having to do the work of two, the left leg being short from old hip disease; and in another (Case 30) from working a treadle. In four cases weakening of ligaments, directly due to acute rheumatism, led to flat foot in the patient on getting about again; these patients' ages varied from eleven to forty-four years, three of them are Cases 5, 29, 34.

Two cases (33, 37) are given as examples of traumatic valgus; but it must be borne in mind that patients with the other forms of valgus constantly say that the pain began quite suddenly whilst standing or walking. This probably marks the onset of the climax of the disease rather than its commencement. In two of the traumatic cases a sprain first weakened the ligaments and flat foot followed; whilst in another a wheel, passing over the foot three months before, destroyed the arch by direct violence.

A case of valgus from ostitis about the head of the astragalus on the inner side, and one from a cicatrix on the outer side of the foot, were also noted.

Weakness of muscles from infantile paralysis was noted occasionally (as in Case 12); but as these cases begin early in life they hardly belong to the present category, although until the child walks nothing is noticed to be wrong as a rule. They illustrate, however, the point already mentioned, that weakening of one

set of "strings" to the bow renders the remainder soon inefficient.

As bearing upon these remarks I must here interpolate a case of true congenital valgus, of which I have Dr. Horrock's report on the state of the muscles.

It was the case of an infant, Lillie B—, æt. 1 year, brought to my outpatients in January 1883, by the mother, as she had noticed the child's right foot persistently turned outwards;—she could easily replace it by manipulation, but the child could not do so by muscular effort. The right limb was well nourished, and there was no evidence of infantile paralysis.

When held so that the feet might touch the ground, the right foot was completely pronated, the child resting on the inner side; it was also flat, but hardly more so than is usual at that age. The left foot, when the child was lying, was natural, but it tended to be everted on standing. Tested with galvanism it was reported "Muscles of right calf do not act to either current. Peronæi act to both currents equally well on both legs. The tibialis anticus and ext. com. digit. of right leg act also well to both currents;" or, in other words, the muscles supplied by the internal popliteal nerve were paralysed.

The *Clinical* aspects of these three groups of acquired valgus differ.

GROUP 1.—*Simple acquired valgus.*

By this is understood some eversion or pronation of the foot, with a variable degree of sinking of the arch. The mildest form of this (Cases 37, 27) is a transition from simple pronated foot to flat foot. In such cases whilst there is marked eversion of the foot, the arch only yields on standing, but when the patient lies down it at once is restored, and consequently the measurements are normal in respect to the metatarso-phalangeal joint.

The degree of eversion is at once judged by viewing the foot whilst standing, from the front; and not only does the inner malleolus seem to overlap, but more of the cushion of the heel appears under the external malleolus than is usual.

The degree of flatness is judged by an inspection of the

inner border of the foot, supplemented by measurement when the patient lies down.

A degree of flat foot on standing, distinctly recognised by eye, may exist without any appreciable difference in the measurement when lying down, and within $\frac{1}{16}$ or $\frac{1}{8}$ in. it is not safe to trust to the tape. In Case 31 the anterior halves of the right and left feet were both $4\frac{2}{3}$ in. and the posterior $5\frac{1}{2}$ and 5 in. The excess of the posterior measurement over the anterior varies of course with the degree of deformity, but in some cases the *posterior excess* stood thus :—Case 9, æt 16, $\frac{5}{8}$ inch in both feet. Case 18, æt. 25, $\frac{3}{4}$ in. left foot only. Case 30, æt. 15, $\frac{3}{4}$ inch in left foot. Case 38, æt 24 (where anterior half in both was $4\frac{3}{4}$), the excess was $\frac{3}{4}$ in. on right side, $\frac{1}{2}$ in. on left. Case 44, æt. 28, $\frac{1}{2}$ in. Case 26, æt. 25, measurement on right side (healthy) $4\frac{3}{4}$ in. for both anterior and posterior parts; on left side $4\frac{7}{8}$ anterior and $5\frac{2}{8}$ posterior measurement. In this last it is to be observed that there was a slight increase in length of the anterior half of the affected side over that on the sound side. An explanation of this will be given under group 3, to which this case properly belongs.

The pain of this condition is usually very characteristically referred (as already mentioned) to below the outer malleolus and across the front of the foot. The reason for this is due not only to compression of the outer edge of the tarsus, from elongation of its inner margin, but to the pressure of the external malleolus against the os calcis; for this ankle pain may exist where there is only pronation and no flat foot, as in Cases 27, 37, 43. At times there is pain about the scaphoid in lieu of the external pain, and more rarely they co-exist in the same case.

Manipulation of the foot reveals more or less convexity of the arch with prominence of the scaphoid and of the astragalus, but the ankle-joint should be freely movable both actively and passively; where this is not so the case belongs to the second group. Even when the foot is quite movable, some degree of rigidity of the peroneal tendons, with generally that of the extensor com. digitorum, is found; but as this rigidity, when excessive, places the case under Group 2, it will be discussed there.

GROUP 2. *Fixed flat foot.*

This condition requires to be clearly recognised, for if treated merely as the cases in Group 1, disappointment will almost certainly follow, while from a malappreciation of its most prominent symptoms, a line of procedure is usually carried out which, to say the least of it, is unnecessary,—I mean tenotomy.

A fixed foot may be more or less flat, but the degree of fixation does not necessarily accord with the degree of flatness. When a fixed foot case walks there is less spring in the gait and more stiffness, and often more pain, than in simple flat foot, and on examining the foot raised from the ground, it will be found set firmly and quite immobile at the ankle. At the same time marked rigidity will be observed of all the external ankle tendons; the peronæi especially, as iron-like bands, being felt behind the fibula. Actual dislocation of these last tendons on to the external malleolus may even occur (Cases 2 and 4). In such a condition nothing is easier than to see an apparent cause for the flat foot, viz. a contracted state of the abductors of the ankle, with consequent extrorsion, and pain. The treatment is equally apparently indicated—tenotomy. But as far as I have been able to see, this conclusion is altogether wrong; nor have I been able to see my way to accepting either the explanation or the therapeutic deductions.

I can see in this condition nothing beyond what one daily sees where a joint is inflamed or otherwise rendered tender, as in early hip disease, viz. a spasmodic or muscular fixation; and Cases 1, 5, 32, illustrate this. Accompanying flat foot at times one sees a diminution in the girth of the calf of the affected side, there being no infantile paralysis or the like; and this helps further in the explanation.

In maintaining equilibrium (since as before mentioned the tendency is for the inner side of the foot to yield) the internal ankle tendons or muscles of the calf have the greater share in the work. Now a muscle will respond up to a certain point of contraction, the heavier the weight put upon it, out after this, it can do no more, and then actual atrophy occurs. Up to a certain (variable) point these internal muscles counteract the tendency to valgus in a man who is, say, overworking and over-loaded; but presently from overwork they yield and as a consequence

even waste. The abductors (peronæi), having lost to a certain measure their opponents, *appear* to contract and become shortened, and present at once a bar to complete restoration of the position of the foot, if even there were no other: they also remain the more powerful set, and will now respond more readily to reflex irritation; whilst if this irritation be permanent they may lapse into the so-called "contractured" condition.

The irritation is that of undue pressure in and between the tarsal bones and more especially in the outer side of the foot; and the nerves supplying these joints are, like the peroneal nerve, derived from the external popliteal, the internal muscles being supplied by the internal division of the great sciatic; thus another link in the chain is found. The degree of pain which the patient complains of is not proportionate to the amount of fixation, as is often seen in early hip disease; fixation of the joint may be marked, while with difficulty can any evidence of pain be obtained.

Fixed foot, partial or complete, has occurred to me in the proportion of about 25 per cent. of all cases of valgus, at all ages, and where various causes have induced the deformity. Like other forms of muscular ankylosis, also, it entirely subsides under an anæsthetic, and the foot becomes as supple as before, and ten days' rest in bed will have the same effect.

If this be the true explanation of fixed foot in acquired valgus—and I cannot doubt it myself—tenotomy is about as rational a general practice as though one were to do it in hip-joint disease for spasmodic fixation, and, still more, it is uncalled for in cases where the foot is not fixed, but merely the tendons are felt to be tight. Yet it is the common orthopædic practice. In a few of my own earlier cases I did it, as I now think, unnecessarily, but in two of these there was actual dislocation of the peroneal tendons, which put another aspect on the matter; and my attention was first directed to the fallacy of the treatment in a case where, the patient being under chloroform prior to tenotomy, I found the foot again movable and the tendons relaxed. I have certainly seen no good result from tenotomy, whilst in one or two cases I think I have traced harm. So impossible is it to commence a rational line of treatment until the abductors are relaxed that, if the patient cannot give the preliminary ten days' rest in bed, tenotomy

might be almost called for, but the gain in time would hardly compensate for other inconveniences, not to say risks.

Prof. Sayre ('Orthopædic Surgery,' p. 62) holds the same views with regard to the ætiology of acquired valgus as those expressed here. He never saw a case, he states, of spastic contraction of the peronei, but thinks all acquired valgus primarily brought about by over use of the internal or antagonistic muscles. He, however, considers the tibialis anticus as the principal muscle in upholding the arch, and finds it in these cases wasted and shrunken from the crest of the tibia. This symptom I have methodically looked for, but never found; and I have failed to trace to it any material influence in these cases, save when I have seen it in a state of spasm at times. That its influence is neutral may perhaps be suggested by its neutral position, for it has its nerve supply as the abductors, but does the work of an adductor.

GROUP 3.—*Flat foot with arthritic symptoms.*

(Cases 15, 18, 20, 26, 36, 38, 40, 44.)

This is the most interesting of the three groups for it is rarely recognised. It is usually mistaken for gout, and the patient is treated accordingly ineffectually, whilst I have heard of excision of the ball of the toe being performed to afford relief.

Some cases of flat foot complain of pain hardly, if at all, in the usual place, but in the ball of the great toe, and if this is examined it is found often in a condition of osteitis. The front half of the foot is often flatter than is usual in even a well-marked ordinary flat foot, whilst the way the great toe lies along the ground in its whole length instead of turning up at the tip is very noticeable. It occurs in about 20 per cent. of all cases. If the other foot happens to be healthy a decided elongation of the anterior half of the affected foot over that of the sound one may be found (Case 26). The pathological condition present is arthralgia, with often an articular osteitis from pressure, but not the usual *elastic* pressure that the normal foot possesses. It usually is seen with marked loss of the arch, but in one case (Case 40) eversion was more marked than flatness. A similar osteitis, but about the internal cuneiform

bone instead of the metatarsal, occurred in one case (Case 20), while in another, a man, æt. 20, who had ostitis of the ball of the toe from direct violence without any valgus, the treatment relieved him as effectually as the valgus cases of this group.

As contrasting with talipes valgus it is very interesting to observe a case of Talipes Cavus which is true "anti-valgus" (Cases 46, 47, 48).

The conditions present are a preternaturally high arch to the foot, the patient walks unduly on the outer side of the foot, and there may or may not be accompanying talipes equinus; the ball of the great toe is prominently projecting on to the sole and approximated to the heel. The toes are all more or less with their first phalanges extended, the great toe especially, and so much is this the case that at times the first phalanx seems to rest on the dorsal aspect of the head of the first metatarsal bone rather than its extremity. In position the toe becomes like cases of true "hammer toe." The plantar fascia (in proportion to the "cavus" present) is more or less tight, and the tendon of the tibialis anticus like a cord starts from under the skin. This muscle is in no state, however, of permanent contraction, for the patient can relax it and has all the movements of the foot. The peronæi cannot, or but very slightly, be thrown into action, but power is relaxed over the muscles passing around the inner ankle. The pain when it exists is a general aching about the ankle.

The cases that have come under my notice, three of which are quoted, have all been due to infantile paralysis, and have been noticed at about ten years old or upwards.

Tested with electricity the peronæi are found paralysed more or less, whilst the tibialis anticus and ext. com. digit. (though supplied by the same nerve) and the adductor muscles act normally, or comparatively so.

The mechanism of the deformity is easy to understand, and its very nature at once shows it cannot be produced by overweight of the arch as in valgus.

If the abductors are paralysed (*i.e.* the peronæi) the adductors (including the tibialis anticus) act unduly; they by contraction both approximate the balls of the toes to the heel, and by taking the strain of the tread off the plantar fascia allow this quickly to

contract, and render the hyper-arching so far permanent. The extensors of the great toe, and of the toes generally, endeavour to extend the toes, but cannot do so fully owing to the fact that the proximal ends of the first phalanges at the heads of the metatarsal bones are kept down in the sole by the plantar fascial contraction. Hence the anterior ends of the first phalanges only get extended, and gradually the whole bone gets pulled on to the dorsal aspect of its respective metatarsal bone, producing the characteristic hammer-like toe. This also explains the prominence of the ball of the great toe towards the sole.

As this condition is not, as a rule, painful, the only treatment is to strengthen the weakened muscles by galvanism, and where "equinus" exists the tendon Achillis should be divided. In extreme cases, however, the foot can be improved in shape, and the great toe can be got into better position (for its being perched up on the dorsum is a serious inconvenience in the matter of boots sometimes) by tenotomy of one or more of the adductor tendons, but especially of the plantar fascia.

The *treatment* of acquired valgus is mainly mechanical, and follows from the explanation that has been given of the disease.

The patient applies because he is in pain, and for no other reason, and his pain being relieved he is—for himself—cured. And this is the most paradoxical part of so apparently rational a treatment, viz. that the pain *is* relieved and the patient *is* cured, whilst the arch of the foot is not proportionately restored. In a few cases, however, there seemed to be improvement in shape, and notably in Case 19. Where there is such complete restoration of the arch there cannot have been more than ligamentous yielding, and the bones must have been unaltered in shape. The pronation or extrorsion was undoubtedly improved, it may be therefore that the misdirected pressure on the tarsus due to this position is more painful than the malposition of the parts due to the falling arch.

All three groups may now be taken together, Group 2 alone calling for special remark. The principle of all successful treatment in acquired valgus is—relieve the over-strained adductors, that they may again recover; offer due opposition to the adductors (peronœi) as nearly like that which they have

lost as possible; relieve the pronation or eversion and the sunken arch by giving *elastic* support upwards and inwards.

Before this can be done all active spasm on the part of the peronæi must be removed. This is not to be obtained by tenotomy, but by putting the patient absolutely at rest, in bed, for five to ten days. By that time all pain and spasm will have gone, and much of the eversion, if not all of it, will have disappeared. The foot is now as in Group 1. In Group 3 manifestly the treatment is to relieve the metatarsal bone of pressure by ordering very easy boots and by supporting the inner border of the foot off the ground, and this is easily done by the same means that are employed to support the arch.

Preliminary treatment with mercury ointment, &c., if there is much inflammation, can be adopted with rest, but I have not usually found it necessary.

With regard to operative measures, apart from tenotomy, they can be but rarely called for, with the improved means of treating flat foot we now possess.

I have known amputation of the front of the foot performed for extreme pain from flat foot; and in four cases (Cases 22, 30, 31, 33) I performed osteotomy at the tarsus, removing a wedge of bone from the inner side, and as far as one could learn this seems a good and sound procedure in cases intractable to other modes of treatment. These four cases receive special notice at the end of this paper.

The dressing that fulfils the above-mentioned conditions is more largely used in America than in England, and was introduced by Mr. Barwell. It will be found fully detailed in his book or in Prof. Sayre's on 'Orthopædic Surgery;' but I have not found all the minutiae he describes necessary to success. I especially refer to the exact imitation of the natural direction of the tendons; indeed, to imitate the direction of the tibialis-anticus tendon across the lower part of the leg will soon result in a sore. His method with strapping is also very tedious to apply, and rather irksome to the patient, so I have had in use for some years a modification of the long-employed inside leg-irons, fitted with an india-rubber "tendon," and a simplified plan for its attachment to the foot.

The original elastic spring-dressing of Barwell is thus made and applied. The things required (for a young adult) are—

1. Strip of tin 7×1 in., *slightly* gutter shaped, with ring or hook soldered into one end on convex side.

2. Strips of strapping: two 2 feet $\times 2\frac{1}{2}$ in. and two 3 feet $\times 2$ in.

3. Tape, needle and thread, and small brass ring size of a shilling.

4. A weak india-rubber door-spring or a piece of stout elastic red tubing, to which hooks have been attached by means of a wooden plug at either end. Its length must vary with its elasticity, but generally $3\frac{1}{2}$ inches will be long enough.

Fix over the shin, in a vertical position, one of the small pieces of strapping, placing one end just below the knee, and lay upon it the tin plate, with the hook uppermost, at the tuberosity of the tibia. Bind it firmly in its place with the two longer pieces of plaster, and then bring up the end of the first piece of strapping, so as to make a loop, supporting the lower end of the tin. The sticky surface of this piece of the strapping will be external, and when applied just over the tin it will cover the hook, so a hole is cut in it to let the hook through; all above this point can be cut away.

The other small piece of strapping is wound round the tarsus, beginning at the sole, and winding it from within outwards round the foot twice; the end which will finally come up from the sole at the instep if now pulled upon will support the inner border of the foot. To this end is now sewn the brass ring, which should lie on about a level with, though in front of, the inner malleolus. A roller is now in the usual way applied round the foot and leg to the knee, a hole being cut to allow the ring with its tongue of strapping to protrude in the sole, and the hook in the leg. This keeps all firm, and if properly applied, the dressing may remain without slipping even for six weeks. The shape and size of the strapping on the foot can be varied to get a pull up more to the front than back of the arch, &c. A forward pull is specially wanted in cases of Group 3. The elastic spring is now attached to the hook in the tin and to the ring below, and thus imitating the adductor tendons at once gives the patient relief.

The strength and length of the spring and other details it is evident must be learnt by experience. It is well to have the spring too strong and too short than the reverse; and if it is

fastened to the ring by a chain or piece of tape, by varying the length of these, the strength of the pull may be graduated.

A hole in the sock is as essential as in the bandage, and one in the upper leather of the boot is advisable to prevent the chafing that happens when the ring is inside the boot.

The sole object of all this strapping on the leg is merely to give a *point d'appui* in the leg for the spring. This is more simply done (and is more cleanly and comfortable for the patient) by using the ordinary inside leg-iron, as for rickets, working in a socket in the heel, and attached below the knee to a collar of steel covered with leather. In this is a stud, over the inner tuberosity of the tibia, to which the spring is attached.

There are two ways of slinging up the foot without the use of strapping.

One consists in a triangular leather tongue being sewn by its base inside and to the outer edge of the boot, the tongue being long enough to cross the sole and turn up to a level with the top of the instep. When the foot is put into the boot in the usual way, if the apex of the triangle be pulled on, the arch is supported and raised. Through a hole in the upper leather the end of the tongue is brought on the inner side, and is there attached by a ring to the spring as before. Thus there is a constant and elastic support provided for the foot.

The other is a sling of soft webbing made thus:—Take a piece of webbing two feet or so long, apply its centre over the *inner* malleolus, bring one end across to the outer side, in front of the ankle, and the other behind it, cross them at the outer ankle and bring them together under the sole and so up to the instep; here attach the ring and proceed as before.

When once the sling has been adapted to the shape of the foot it can be sewn, so that in future it will slip on and off like a collar.

All this apparatus need only be worn in the day, but the strapping if used cannot be removed, though the spring can be unshipped at night.

It need hardly be added that where the object is to raise the foot, the boot must be large enough to allow of the movement. This apparatus in tight boots cannot answer.

For treatment from the first, sole plates have not succeeded in my hands, nor any of the ways of pushing up the arch by

cork, &c. by a dead pressure. The *elastic* lift of the spring it is that gives the success to this mode of treatment, though as after-treatment—when all pain is gone—sole plates are all very well.

I have tried, and it promises to succeed, an elastic sole plate. It consists of a steel plate, flat like a cork sole, which fits into any boot, having playing on it a raised curved steel spring plate made somewhat to the shape of the foot. This is rivetted to the first plate behind, the front part alone being movable, so that it yields with the tread and resumes its shape afterwards.

The results of this method of treatment in my hands have been very good. Almost without exception has immediate relief been given. In many of the cases the patients were lost sight of as soon as they felt relief, and this was especially the case in hospital patients who supply two thirds of the total number. Some of these, however, I have kept up with; and these together with the remainder from private sources enable me to say that the spring-dressing treatment (by the strapping or other method) has given such perfect relief to pain as to deserve the name of a cure, and in almost every instance when this dressing was discarded nothing further was required in the way of after-treatment.

The length of time for treatment has been from one to six months.

The four cases that have been alluded to in which osteotomy was performed are Nos. 22, 30, 31, 33. They came under my care before I had adopted the elastic spring dressing, and the immediate cause of the idea of operation being entertained was the suggestion made to me by Mr. Cock—who kindly saw one of them with me—that amputation was the only means of removing the pain. Arguing, as I have already shown, that the increase of the inner margin of the foot must cause compression of the tissues in the outer, I thought that by removing the keystone of the arch I might both remove the cause and the pain. In four bad cases, therefore, I removed the scaphoid, and in one of these (Case 22) sawed across the tarsus as well. As in all (with this last exception) the operations were alike, I will describe it here once for all. A vertical incision was made at the inner margin of the foot over the scaphoid, and the soft parts being turned back from off it, it was seized with the lion

forceps and partly by traction and partly by leverage with a blunt dissector, ligaments being previously divided, it was forced from its position. The limb was then put in an ordinary back splint, with foot piece, this last, however, being in two pieces, united by a dish joint, so that the front half could be adducted on the posterior, and this adducted position was maintained during convalescence as much as possible.

In no instance did anything untoward occur, and the patients all recovered, but the result of each is mentioned with the history of the case. Plate II, taken from casts of Case 31, showed markedly the improved shape of the foot.

In two cases the patients were not seen after their discharge from hospital, and, therefore, it is impossible to speak certainly of results, but the fact that they left without pain and did not return for treatment suggest success. In the remaining two enough was seen afterwards of them to allow of the reality of the relief given being tested.

No. 1. Chas. L—, æt. 17. May 14, 1879. Engineer. One year's history. Overstanding. Pain below outer ankle and across front of joint, very severe at times. Left foot affected. Calf measures five eighths of an inch less in circumference than on right side; no special wasting of anterior tibial muscles to be discovered. Pressing centre of tarsus between thumb and finger from below upwards causes pain, and the peronæi and extensor communis digitorum are at once thrown into spasm. The slightest attempt at adduction of the front of the tarsus has the same effect. The tibialis anticus remains unaffected. The arch is completely lost and the tarsus pronated; fixation half. A healthy lad, but hands and face red and cold; no cardiac bruit.

Treatment.—Spring dressing during five weeks.

Result.—Last seen June 16th, 1879. "Walking comfortably—no pain."

No. 2. Mary Ann S—, æt. 14. May 26th, 1879. Works in the fields, always standing. Mother noticed her walking uncomfortably for a year, complaining of increasing pain, so bad the last three weeks that she could not get about. Left foot affected; complete fixation of foot; rigidity of tibialis anticus, extensor communis digitorum, and both peronæi;

these last dislocated on to external malleolus. Marked pronation, but only slightly flat. Cannot be forcibly adducted. Left foot a quarter of an inch in total measurement longer than right. Circumferences of both calves equal.

Treatment.—Commenced as in-patient July 8th, 1879, by tenotomy of the peronæi. On 17th she left, walking without pain and wearing spring-dressing.

Result.—Relieved.

No. 4. Eliza B—, æt. 17. In-patient. 11th April, 1879. General servant, always on her feet. In November, 1878, returning from a walk, felt severe and sudden pain in left ankle, rendering the leg “powerless,” and which has incapacitated her till now; getting worse. Complete fixation of the left foot, which is also pronated and flat. Peronæi are rigid and dislocated on to the external malleolus, to which she ascribes the sudden pain in November last.

Treatment.—On May 5th tenotomy of the peronæi. Discharged with spring-dressing on June 9th, no better (due apparently to apparatus not acting). Dressing reapplied June 23rd. Walks without pain. July 21st.—Dressing changed for simple strapping, which was continued till October 20th.

Result.—Since June had no pain and walked quite well. Till September slight fixation of foot on standing, when this symptom disappeared. Shape of foot not altered, still flat. Dismissed cured.

No. 5. Mary S—, æt. 44. In-patient. June 16th, 1879. A washerwoman, always standing. Cannot work now on account of severe pain in both ankles, coming on on getting about after rheumatic fever two years and a half ago. Has noticed her feet altering in shape during this time. Marked flat foot, but left side worse. Peronæi on both sides dislocated forwards. Complete fixation, rigidity of the peronæi and extensor communis digitorum. Foot extremely tender on pressure, and peronæi then felt to quiver by reflex action. When standing rests on outer edge of feet as much as she can.

This patient did not remain in for treatment.

No. 9. Chas. M—, æt. 16. Butcher boy, carrying meat. A year's history. Has marked flat foot, but no fixation. The

peronæi are very rigid. All the pain is confined to the region of the scaphoid bone, below inner ankle. Measurement showed posterior half of foot five eighths longer than the anterior.

No treatment, he only came once to out-patients.

No. 11. Nellie G—, æt. 17. Out-patient. 31st May, 1880. No occupation, and no special cause, but is a weakly girl. One year's history of pain in both ankles, the left being the worse. Both feet flat, but not fixed; pain situated below the external malleoli.

Treatment.—Spring-dressing to both feet, continued till August on the right side and September on the left. All treatment then suspended. To wear boots with inside irons for a time.

Result.—Complete relief from pain from the time of using the dressing, unless the springs went wrong. Before finally giving it up it was gradually rendered less and less effectual, but yet there was no return of pain. Dismissed walking well without pain, and the right foot seemed to have recovered its arch somewhat.

No. 12. Victor C—, æt. 8. Out-patient. June, 1880. Has old infantile paralysis of left leg, since birth. Always had foot flat and pronated. Walks completely on inner side of foot, where also the scaphoid and astragalus make a large convexity. Has scarcely any power over the foot, all the ankle muscles being equally affected. No pain.

On June 28th treatment with the continuous current thrice weekly commenced. August 9th.—Leg fatter, firmer, and warmer, all the helpless “flabbiness” of the foot gone, having good power over the ankle; foot as flat as before but not so everted (pronated). May, 1881.—Walks well. Complete restoration of power in the muscles acting on ankle, but foot still flat as before. Cease treatment.

No. 13. Frank H—, æt. 11. Out-patient. November, 1880. A potman, and always standing. Had pain first six months ago. Has marked flat foot, with some pronation. Foot not fixed. Pain under outer ankle and across front of joint. Right side only affected.

Treatment.—Spring-dressing from December 1st, 1880, and

continued till April, 1881. Within two weeks of commencing treatment all pain ceased, and when the treatment was discontinued there appeared also to be some improvement in the shape of the arch, and the pronation was less.

No. 14. John P—, æt. 15. A paper-ruler and standing constantly. Three years' history of pain in both feet. Had from time to time had his ankles strapped, affording him temporary relief. Is a pale, anæmic lad. He was admitted into Lazarus on November 29th, 1880. Both feet flat and painful and pronated. Pain of left foot under inner ankle, of right foot under outer ankle; both feet quite fixed, right foot worse. Measurement of right foot ten inches and three eighths, of left ten, in length.

Treatment.—Kept in bed from November 29th to December 14th, when all fixation and pain disappeared and the ankles were freely movable, passively and actively. Spring-dressing applied on December 14th, and he was dismissed on December 17th walking comfortably without pain. On January 2nd, 1881, he walked twelve miles, being seven hours on his feet without discomfort or pain. All treatment stopped on March 28th, 1881. Completely relieved.

No. 15. James L—, æt. 17. Out-patient. 3rd January, 1881. A packer, and always on his feet. After a day's work gets great pain in both ankles, under both malleoli, and in the balls of the great toes. His muscular system is ill-developed, and both feet are flat but not fixed.

No treatment, as he was only seen once.

No. 17. Ann E—, æt. 13. Out-patient. 1st January, 1881. For two years has had pain across front of ankle and on inner side of right foot. The right foot is flat but not fixed. The left leg is short from hip disease, and she wears a high-heeled boot and thus has thrown more work upon the affected foot. On January 17th the elastic spring dressing was applied to the right side, and reapplied from time to time as requisite, until May 2nd, when she continued free from pain, as she had been since the treatment was commenced.

She was under treatment till May 30th, 1881, when she ceased to attend, being quite free from all pain.

No. 18. Alfred S—, æt. 25. Out-patient. January, 1881. A labourer, and standing constantly. For one month had great pain in ball of left great toe and beneath outer ankle. He has complete flat foot with some pronation. The metatarso-pharyngeal joint of great toe is red, tender, inflamed. The toe lies markedly flat, not tending to rise from the ground at all. No fixation. Posterior half of foot is nearly three quarters of an inch longer than the anterior.

Treatment.—Spring-strapping dressing applied on January 24th. By February 28th he was at his work, free from pain, and on March 21st he ceased to attend, having lost all his subjective symptoms. The inflammation of the toe had quite subsided.

No. 19. Alice B—, æt. 14. Out-patient on February 28th, 1881, for “weak ankles.” She had no pain, but her friends observed her mode of walking. Both the ankles seemed to give in so that she had what is commonly termed in-growing ankle. There was no pain. Both feet were flat and rather fixed; the right being the worse. Marked pronation (or eversion).

Treatment.—The elastic spring dressing was employed for three months, and she much improved; but as the treatment could not be carried out thoroughly, she was ordered to wear high boots with steel plates in the soles. She wore these twelve months. She was seen last in February, 1883. There was no flatness now at all, but nearly the same eversion, or as the friends put it, “ingrowing and weak ankles.” The heel was very movable from side to side by the hand. No pain. The girl was wearing narrow-soled and high-heeled “fashionable” boots. She was advised to wear low heels and broad soles and to have the elastic spring apparatus fitted to the boots.

No. 20. Thos. P—, æt. 40. Out-patient. 28th March, 1881. A cooper, and most of his work is planing, his weight coming down upon the left foot. For six weeks has had pain in middle of left foot, especially in the evening. The right foot is normal. The left foot is flat and pronated, and there is swelling, heat, and redness just over the internal cuneiform bone, indicative of ostitis.

Treatment.—Tonics and local application of Ung. Hydrarg.,

under which in two months the pain (ostitis) subsided. He then ceased attendance.

No. 22. Kate M—, æt. 12. A servant. For five years before had been in an industrial school and had lived badly. All that time had suffered from pain in the feet, which came on suddenly but has gradually increased. She was under my care as out-patient from March to November, 1878, being strapped and wearing a steel sole plate. Both feet were quite flat, and the right rather fixed; the right was the worse. The pain was referred on both sides to below the outer malleoli. She became an in-patient in November, 1878. Both feet were flat and ex-torsed; the right showing three-quarters of an inch excess in measurement over posterior half; the left (?). The right foot was put up in an iron splint and plaster-of-Paris bandage, and she walked out relieved on November 22nd. In January, 1879, she was re-admitted again suffering in both feet, the right being the worse and more fixed than the left. On January 9th, the peronæal tendons on the right side were tenotomised and the foot was strapped so as to support its inner margin. By January 31st there was no relief, and so the scaphoid was removed, and the tarsus was afterwards completely divided across (and the foot placed in the splint already described). On February 22nd she was sitting up and on the 25th walked about the ward without any pain. She was discharged wearing a plaster-of-Paris stocking, and subsequently a Martin's bandage on account of œdema and for support. In August, 1879, the note made was, perfectly well; to give up the india-rubber bandage. In September, 1879, had still no pain. She was next seen in July, 1881, when these notes were made (Out-patient notes, 202 F.): "Last four months pain in sole of foot. Nothing to account for it. Till then had been quite well since the operation; has had no return of her flat foot pain under the external malleolus. When standing, the inner edge of the foot is raised, save the heel and ball of toe, and she walks with a stiff springless action." And again on October 24th, 1881: "Foot seems all right barring stiffness. There is, she says, pain that prevents her walking, but I doubt its being genuine knowing the way she behaved in hospital. She has the inner side of the foot well raised on standing."

No 24. David M—, æt. 18, Out-patient. June 12th, 1882. Works at a gas factory and is constantly standing. For eighteen months has had pain below outer ankle, for which he now sought relief. Both feet are very flat and much pronated, but not fixed; pain beneath outer ankles. The arch is represented by a marked convexity of the scaphoid and head of astragalus. Rigidity of the peronæi and of the extensor communis digitorum.

Treatment.—Spring-strapping dressing applied on July 3rd, and on July 17th he was back at work without any pain. The dressing was worn until November, and on the 27th of that month this note was made: "There appears some arch returning, has no pain."

No. 26. James B—, æt. 25, came first on 23rd October, 1882, stating he had had rheumatic fever five years ago, and did not walk for eighteen months on account of pain in knees and legs; after that he returned to work. Three months ago in a carriage accident he caught his foot in the harness and had it "wrenched;" it swelled up immensely; when the swelling subsided the foot was altered in shape as at present. Has not been able to work since, but before the accident his work involved much standing.

His present condition is:—Left foot very flat with bulging of astragalus on inner side; so much pronation or eversion that on standing the leg seems quite to overshoot the ankle in an inward direction, has great pain in the ball of the toe which travels up the front of the leg; there is pain also beneath the external malleolus. The inner border of the foot is very flat on the ground, and markedly so from the internal cuneiform bone forwards, the ball of the toe is not red nor tender to touch. The foot is not fixed, yet does not move very readily. The right foot is normal. For two weeks he was treated with strapping as out-patient and was admitted on November 15th, so as to have suitable apparatus fitted, and to give him rest in bed. It was found on admission that the foot was partly fixed; the peronæi and ext. com. digit. were very tense. Measurements, right foot anterior half $4\frac{3}{4}$ in., posterior half $4\frac{3}{4}$ in., total $9\frac{1}{2}$ in.; left foot anterior half $4\frac{7}{8}$ in., posterior half $5\frac{1}{4}$ in., total $10\frac{1}{8}$ in. No wasting of muscles of leg.

Treatment.—Rest in bed for three days caused entire subsidence of the spasm of the abductor muscles. The spring appar-

atus with inside iron, and sling for the foot were fitted, and he was discharged on November 24th, walking without pain. He reported himself some time after, and was still without any pain.

No. 27. Florence I—, æt. 13. Out-patient. 8th January, 1883. For three months had had pain below outer ankle on right side. When lying down only slight pronation is noticed, but when standing this becomes marked, and partial loss of arch of foot appears. Complains only of the pain as above. The foot is fully fixed, and the peronæi and extensor communis digitorum are very tense. No wasting of muscles. The patient is very healthy in appearance, and lives at home, does no work, and there is no evident immediate cause for the deformity.

Treatment.—Nil; only seen once.

No. 29. Frank E—, æt. 14. Was in-patient from 7th February to 23rd February, 1878. Two years before he had had acute rheumatism, and six months later had pain in the right foot. This as he got about gradually increased, and for the last four months has been much worse. Has obtained some relief as out-patient by having ankle strapped. On examination there is marked eversion of the right foot, with rigidity of the peronæi and extensor communis digitorum. The arch is sunken, and the astragalus forms a projection towards the sole.

Treatment.—On February 12th the rigid muscles were divided, and an outside splint applied. On 17th the elastic-spring dressing was put on, and he was discharged, walking comfortably, on February 23rd. Not seen since.

No. 30. Geo. K—, æt. 15. For two years was acting as a milk boy, and then for five months has worked a lathe treadle about eleven hours a day; the last four months has had to give up on account of pain in both feet, and he noticed them getting flatter. After two months from the pain commencing it increased so that he could barely stand in the evening. He became in-patient on 29th May, 1878. Both feet flat and everted; no arch at all to left foot (the worse), and but slight arch on the right; posterior excess on left side three quarters

of an inch. On May 31st tenotomy of the peronæi on both sides was performed, and the feet eventually were placed in plaster-of-Paris stockings. On June 7th the elastic-spring dressing was substituted, and on June 17th he was discharged walking comfortably. A week later he was readmitted in as much pain as ever on the left side; so on July 3rd the scaphoid and part of head of astralgus were removed. On August 29th the wound had healed and he was about on a chair. On October 2nd he was discharged, walking well, without any pain.

No. 31. Adelaide R—, æt. 17. In-patient from 25th November, 1878, to June 12th, 1879. Two years before had pain in right foot come on whilst standing, and has during that time been attending as out-patient, having foot strapped, &c. Pain (slight) commenced in left foot six months ago. Right foot flat and everted; anterior half of both feet $4\frac{2}{3}$ in.; posterior half of right one $5\frac{1}{2}$ in., of left 5 in. Pain situated over all the sole and at outer ankle. Complete fixation. Right calf 11 in. in diameter; left one $11\frac{1}{2}$ in. Can walk about a mile, no further. On December 7th, 1878 the scaphoid was removed on the right side, and on January 3rd, 1879, she was up, with a Martin's bandage on. On January 12th she was discharged, wearing a plaster-of-Paris stocking. She has not been seen since then; the result is, therefore, conjectural, but she was free from all pain when she left, and in all likelihood would have come again had it returned. (See Pl. II, figs. 1, 2.)

No. 32. Amy C—, æt. 17. Was admitted into hospital on December 5th, 1878. She stated that three years before, whilst standing, she felt something slip in her ankle, and she was immediately thrown down on the floor, "having no more use of her left foot," and it has been painful ever since, being worse some times than others. She is a servant, and stands about. She had applied for relief at various hospitals, but had not followed out the treatment. Examined it was found that the left foot was flat and very extorsed (pronated), and that she walked quite on the inner side. There was rigidity of the peronæi and extensor communis digitorum, and the toes were drawn up in extension. The foot was fixed. When being examined under chloroform it was observed that as she became narcotised

irritation of the right foot produced no effect, but in the left foot marked spasm of all the muscles supplied by the external popliteal nerve occurred on touching the sole.

Treatment.—On December 8th the peroneal and external communis digitorum tendons on the left side were divided, and a plaster bandage, encasing an iron splint with foot piece, was applied. On January 6th, 1879, she was discharged wearing this, and was free from pain.

Readmitted on November 5th, 1879, for pain in the right foot, the left remaining well. On the right side there was well-marked flat foot, with projection of the astragalus and rigidity of the abductor tendons. The *treatment* was just as on the other foot, and she was discharged free from pain on December 9th, 1879, wearing splint and plaster-of-Paris bandage.

No. 33. Sydney W—, æt. 15. In grocery business, always standing. Three years before coming for advice sprained his right ankle, which six months later began to “project,” and there was great pain on walking, which has continued to the present. The patient, on admission November 11th, 1878, is tall and strong for his age, but his legs are markedly non-muscular and flabby. On the left side the foot is quite flat, everted, and the scaphoid and astragalus make two well-marked tubercles on the inner side. There is tenderness on pressure over these bones. On November 16th part of the left scaphoid and head of the astragalus—as a wedge—were excised, and the peroneal tendons were divided. On December 18th the wound was quite healed, and he was dismissed on December 20th wearing a plaster stocking. Seen last in March, 1879. He wore the plaster stocking for four weeks, and since then nothing at all. Has had no pain, and gets about as usual. The foot is still flat, but not everted, and the posterior half is half an inch in excess of the anterior. (Unfortunately the measurements before operation were not entered in the report.)

No. 34. John H—, æt. 30. A blacksmith. Was seen first on June 3rd, 1881, on account of pains in his feet. Twelve years before he had had acute rheumatism, and again fifteen months ago, since which time he has had pain in his knees and

ankles, and owing to this and their extreme weakness he can hardly walk.

Both knees are weak, in condition of valgus, and yield when he walks; both feet flat and much pronated (extorsed), and very painful.

Treatment.—He was kept in bed from 3rd to 20th January taking salicylate of soda, and then the elastic spring dressing was applied to both feet and a short jointed outside splint to the knees. This dressing he wore at a convalescent home till March 8th, when he reported freedom from pain and ability to walk two miles along the sea beach. The dressing was reapplied to the feet; the knees were straight.

He was last seen in May, 1881, and reported to be perfectly well.

No. 36. Mrs. H—, æt. 43. In the spring of 1882 had much standing about and gradually suffered from pain in balls of both feet which increased in degree, until at last she could only limp about, and had taken to soft felt boots as the bones were so very tender. She had been lying up for weeks without any permanent benefit, and also, with the same result, had been treated for gout. When examined both heads of the first metatarsal joints were red, swollen, and very painful, the veins over them being well marked. Both feet were slightly flat, and the right one especially very pronated, the anterior half of the inner side of the foot being particularly flat on the ground. On slinging up the feet with a bandage the patient at once stepped without pain.

Treatment.—The elastic spring apparatus with ankle sling. In three weeks the patient could walk a little with scarcely any pain, within two months walked two miles and felt nothing.

No. 37. Frank S—, æt. 38. Seen first on 19th December, 1882. Six weeks before sprained his foot getting out of a dog-cart, and he has now marked extorsed (pronated) foot, but only flat foot when standing; when lying down the arch returns. Measurements are normal on the inner side of the foot. Pain below outer ankle and along front of the joint. He was fitted with the iron support, foot sling, and spring, and within a week was walking nearly free from pain. The last

report, received six weeks after first being seen, showed complete freedom from pain.

No. 38. Edward S—, æt. 24. For three years had had gradually increasing pain in ball of right great toe, and right foot at same time became flatter. Walks much and thus aggravates the pain.

When first seen in June, 1880, the right foot was flat, and the measurements were, anterior half four and three quarter inches, and posterior half five and a half inches. The ball of the toe was red, swollen, and tender—(gouty looking)—and the joint stiff.

Gets relief in walking from throwing himself on the outer edge of the foot, as the wearing of the sole proves. No pain at outer malleolus, foot not fixed, but markedly everted. Left foot similarly affected in a very slight degree, its measurements being, anterior half four and three quarters, posterior half five and a quarter inches nearly.

The elastic spring dressing was applied directly, and after six weeks the apparatus with the iron rod was used in lieu of the strapping, to both feet. This was continued till January, 1881, or for six months, during which time, unless the springs failed, there was no pain at all. By Christmas, 1880, he could walk two or three miles with ease, and in January, 1881, left off everything, being relieved of all his symptoms. There was some reappearance of the arch in the right foot, and all the eversion vanished. For two years he continued to wear a boot with stiffened leather under the instep, and with it walked about comfortably.

No. 40. Henry M—, æt. 22. First seen in March, 1881. A year before he began to have great pain in the ball of the right toe. He knew of no cause for it himself, it came on only after walking and went away on resting. He is a great walker and can now do his ten miles, but in pain. Is accustomed to throw himself upon the outer edge of the foot to get relief. The foot, on examination, seems slightly flat with swelling of ball of great toe, and tenderness on manipulation, but no redness.

Treatment.—He was fitted with the spring apparatus and leather tongue in sole of boot, to which the spring was attached.

On June 22nd, 1881, I noted "has worn boot with spring apparatus till two weeks ago; had no pain, and has had none the last fortnight also."

No. 43. Miss N—, æt. 14. A weakly girl, stoops much, and one of a family in which is a history also of flat foot and lateral curvature. Was first seen in July, 1882, for pain under the outer malleolus of the right foot. On examination neither foot had a high arch; both were pronated (extrorsed), especially the right, but the measurements were exactly normal, the metatarsal-cuneiform joint being four inches and a half alike from tip of toe and heel.

She was ordered the spring apparatus with leather tongue in boot. Further history not obtained.

No. 44. Miss H—, æt. 28. May, 1880. Three years' history of pain under arch of foot and beneath outer malleolus, followed by severe pain in ball of great toe. Ascribes it all to over-walking in a mountainous country. The ball of the toe at times is very tender, inflamed, and œdematous; when this is not the case she can walk fairly well for short distances on level ground, but uneven walking or stairs at once bring back the pain, which generally subsides at night when in bed, but not always. There is marked flat foot with pronation (extrorsion), but no fixation. Posterior measurement four inches and three quarters, anterior measurement four and a quarter. She has worn steel plates and very loose boots, and has been treated for gout, but all to no purpose.

Treatment.—The elastic-spring dressing, &c., prescribed, but nothing more was heard of the case.

Pes cavus.

No. 46. Alice S—, æt. 16, out-patient Nov. 27th, 1882, has old infantile paralysis affecting the right leg, which is wasted and smaller than the left. Complains of general aching pain about ankle. Has much standing and walking, the right leg is colder than the left, and Dr. Horrocks kindly furnished the following report:—"muscles of right (lower) limb act as readily

to both currents as those of the left, but much less strongly, except the peronæi on the right side which do not act at all to galvanism and only very slightly to a strong Faradic current." The foot is extremely arched and the patient walks on the outer edge, the plantar fascia is contracted, and the tibialis anticus and posticus she can throw into fairly powerful action. There is the usual hammer condition of the great toe; no talipes equinus.

Seen last on December 18th, 1882, after being regularly galvanised. She then walked better and the limb was recovering warmth and power.

No. 47. Robert K—, æt. 12, outpatient in January 1883. From infancy he had wasting and coldness of the right leg; and now has marked talipes cavus. The arch of the foot is very high, there is commencing talipes equinus, and he walks unduly on the outer side of the foot. Usual hammer shape of great toe; slight contraction of plantar fascia. Has power over the tibial muscles but not over the peronæi.

To be treated at first with galvanism.

No. 48. Alice S—, æt. 18, in-patient from January 10th, 1876, to 16th February, 1876. In July 1875 she was first noticed walking on the outer edge of the right foot, which is colder than the left. Parents knew of nothing wrong prior to the date given. There is marked talipes cavus with great spasm of both the tibial muscles. Tested with galvanism it was found that thirty-five cells which produced powerful contraction on the left side, had no effect at all upon the peronæi of the right side. The other muscles responded to fifty-five cells on right side but to thirty-five on left. Thus the right peronæi were practically useless, and the other muscles weak as compared with the left side.

Treated with galvanism till February 16th when she was discharged well.

DESCRIPTION OF PLATES.

PLATE I.

FIG. 1.—*a*. An outline of the bones of the foot and soft parts, illustrating the centre joint on the inner side of the foot (p. 440). *b*. A similar outline of a flat-foot, showing the advance of the centre joint (p. 441).

FIG. 3.—The leg-iron supporting the steel collar to which the india-rubber spring is attached (p. 456).

FIG. 4.—The sling used to support the arch in lieu of strapping (p. 456).

Fig. 2.—The sling applied.

PLATE II.

FIG. 1.—Front view of the right foot (standing) of Case 31 before treatment.

FIG. 2.—The same after excision of the scaphoid. The compactness and improved position of the foot are well shown as compared with the flatness, and eversion of Fig. 1. Both these figures are drawn from casts.

FIG. 3.—The side and front view of the foot of a lad, æt. 18 (K. 231). The projection of the astragalus and scaphoid bones destroying the arch are well seen in the former, and the extrorsion in the latter. There was one year's history of pain and he was always on his feet. The total measurement of the foot was $10\frac{1}{2}$ in., the front half being $4\frac{1}{2}$ in., the posterior half $5\frac{3}{4}$ in.

1



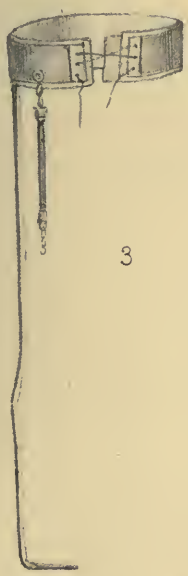
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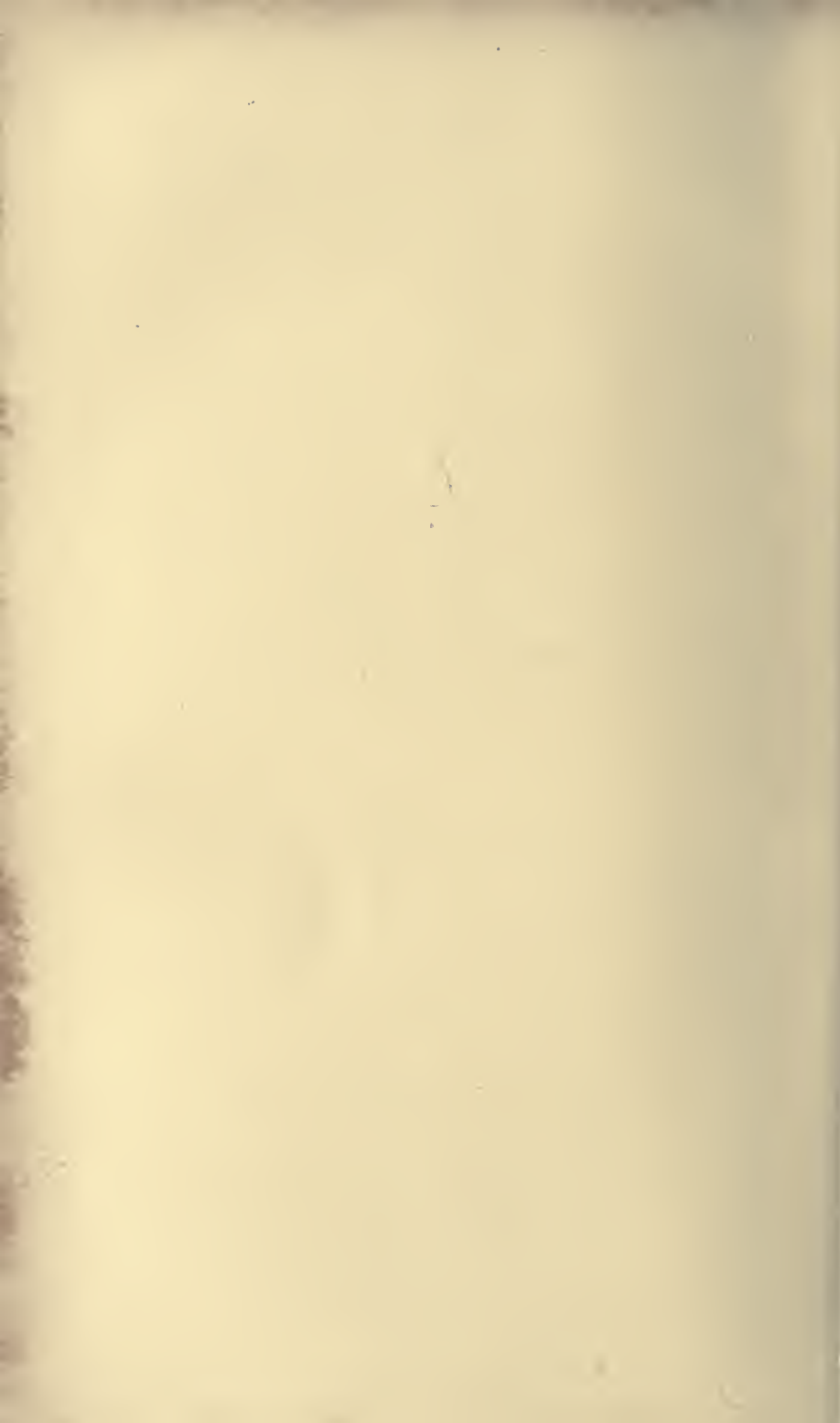


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LEAD-POISONING.

BY THOMAS STEVENSON, M.D.

THE subject of lead-poisoning, trite as it may at first sight appear, has recently attracted fresh attention. First, the condition of the workers in white-lead factories has caused much commiseration; but into this form of lead-poisoning it is not my present purpose to enter. Secondly, the conditions under which drinking water acts upon lead have acquired new significance from the facts elicited both at a trial at the last Leeds Summer Assizes, and at an inquest held at Keighley in October last. And, thirdly, a conviction for murder took place at the last December sittings of the Central Criminal Court (*Reg. v. Louisa Jane Taylor*), in consequence of an old woman's death having been hastened by the homicidal administration of lead acetate (sugar of lead). This is the only case, so far as I know, where, in this country, death has resulted from the homicidal administration of this salt. Cases of poisoning by sugar of lead, taken with suicidal intent, are common enough, with serious results only in exceptional cases.

A solicitor practising in Huddersfield, and living in the outskirts of that town, was attacked about the middle of 1881, first with violent colic, and ultimately with wrist-drop and other symptoms of lead-poisoning. The disease advanced to such an extent that he lost almost entirely the use of his arms, and his brain became affected. He subsequently partially recovered under medical treatment; but even after a year

had elapsed he was still unable to dress himself, though he had partially recovered the use of his limbs. The fact that the patient was poisoned by lead was indisputable, and the question now arose as to how he had imbibed the poison. Naturally suspicion fell upon the water supply derived from the mains of the Corporation, which, as in most of the large northern towns, caters for the supply of water to the inhabitants.

The circumstances which led to the pollution of the water with lead are of extreme interest, and were greatly elucidated by the experiments of Drs. Odling, Crookes, and Tidy, and those of Messrs. Allen, Fairley, and Jarman. The Huddersfield water supply is very soft and pure; but like the waters supplied to Leeds, Bradford, Halifax, Sheffield, Batley, and Manchester, the Huddersfield water, if allowed to rest in leaden pipes for twelve hours, takes up appreciable quantities of lead. The town of Huddersfield derives its water supply from several similar springs; and the patient above referred to had his supply from the Blackmoorfoot dam, which affords the purest supply in the town. The water in the iron mains was found to be free from lead; but after passing through the leaden supply pipes to the patient's house, quantities of lead varying from 0.01 to 0.84 grain per gallon were found to have been taken up.

An action for damages for injury to health was brought by the aggrieved person against the water company (the Corporation), and heavy damages were recovered, subject to a point of law as to whether the company was bound by the general Act governing public water companies to supply pure water into the dwelling house, or whether the company fulfilled its legal obligation by supplying pure water in the mains. It appears that the Huddersfield Corporation has power by its bye-laws, framed under its own local act, to define the material which may be used for service pipes, which belong to the owner of the house and not to the Corporation. Leaden service pipes are, however, permitted by the bye-laws. The decision of the Superior Court was, that the water company fulfilled its legal obligation to supply pure water by supplying pure water in the mains, and was not bound to maintain it pure in the service pipes.

But why should the Blackmoorfoot dam water act so largely upon lead? It is not all pure soft waters which do so; and

this water was not only poor in saline constituents, but it was also almost destitute of organic matters; and the presence and not the absence of these is usually supposed to increase the solvent action of water upon lead. The explanation is as curious as interesting, and has an important bearing upon the water supply of towns generally. The Huddersfield water is destitute of earthy carbonates, and is indeed feebly acid in its reaction to delicate reagents. This activity is perhaps not due to the actual presence of a free acid, but is attributable to the existence in the stored water of an acid ferric salt (persalt of iron). It is well known that soluble ferric salts are acid in their reactions.

Now, the Blackmoorfoot dam at Huddersfield receives certain ferruginous springs, containing iron in the state of ferrous salts (protosalts of iron), and these ferrous salts when exposed to air in solution become oxidised, and split up into a basic ferric salt which is deposited, and an acid ferric salt which remains in solution. It would be impracticable, even if desirable, to deprive such a water of oxygen, so as to prevent its action upon lead. Mr. Jarmain has, however, recommended the addition of lime-water as a preventive to corrosion. In connection with this subject it may be mentioned that the soft water supplied to Glasgow corrodes the plates of steam boilers, and this corrosive action is attributed with reason to the oxygen dissolved in the water. Mr. Jarmain's proposed remedy deserves at least a careful trial.

At the trial at Assizes there was a conflict of opinion among the chemists as to the effect of the acidity of the water upon leaden pipes. It was assumed that there was free sulphuric acid present, though of this there was no conclusive evidence; and Drs. Odling, Crookes, and Tidy, held the opinion that sulphuric acid, if present in small quantities, must tend to protect the pipes from the action of the water, by forming a thin layer of the insoluble or very sparingly soluble lead sulphate. Mr. Allen took a different view, and held that sulphuric acid would cause corrosion. It is probable that the action of an acid ferric sulphate would be pretty much the same as that of free sulphuric acid. The acidity of the water was small—about the equivalent of one-eighth of a grain of oil of vitriol per gallon.

Since the trial Mr. Allen has investigated the subject experimentally, and finds that this small quantity of sulphuric acid notably increases the solvent action of a soft water upon lead. The sulphate of lead is more soluble in water than the hydrocarbonate (white lead), and yet it is well known that the latter dissolves in water to such an extent as to render it poisonous.

Not long after the civil trial at Leeds an inquest was held at Keighley in Yorkshire on the body of a man who had died, as was surmised, from lead-poisoning by water-supply. Here, again, the local authority, the Keighley Local Board, supplies the town with water. I gather from the reports of the inquest, and a report published by Dr. Dobie, one of the medical men who attended the deceased, that the latter, a man forty-two years of age, was in the habit of drawing from the tap, and drinking, a glassful of water every morning early. The acid water, after standing all night in the pipes, was found to have taken up 0·61 grain of lead per gallon, a highly poisonous amount. Two years before his death the deceased suffered from constipation and colic, and he had a blue gum-line. In the spring of 1882 he was affected with dyspepsia and sleeplessness, from which he recovered under change of air, but the symptoms returned when he was again at home. In the following August his disease was aggravated, he had headache, sleeplessness, and at length delirium, paroxysmal "dyspepsia," frequent vomiting, colic which was increased by pressure, obstinate constipation, convulsions, and a blue gum-line which disappeared after the use of potassium iodide for eight days, and three days before his death, which occurred on August 26th. He had some use of his hands up to the time when he became comatose, twelve hours before death, and there was no pronounced wrist-drop. When the arms were raised the hands dropped, but could be raised by an effort of the will. There was a peculiar fœtor of the body. The body-temperature never exceeded 97·7° Fahr. The convulsions and colic alternated. The urine was albuminous. There was not at any time an ammoniacal or urinous odour of the vomited matters.

The post-mortem examination showed a dropped left wrist (?), with no extreme wasting, and the extensor muscles of the fore-

arm were apparently healthy. The colon was contracted and thickened in its ascending and transverse portions, with enlarged follicles in the thickened parts. The kidneys showed granular degeneration, but it was thought not to be so far advanced as *per se* to account for death. The heart was hypertrophied, and the left auriculo-ventricular opening was enlarged. The brain appeared healthy, and there was no effusion into the ventricles. Mr. Allen found doubtful traces of lead in the kidneys, none in the brain, and in the liver and spleen together one fourth of a grain.

Drs. Dobie and Jack were of opinion that the man had died of lead-poisoning, to which Mr. Allen's analysis also pointed; and this gentleman—a chemist and not a medical man—was of opinion that a patient suffering from lead-poisoning might be freed from the metal in a fortnight by the administration of potassium iodide. Dr. Tidy gave evidence at the inquest strongly antagonistic to the theory of lead-poisoning, though he declined to assert that lead had nothing to do with the death, and the jury returned a doubtful verdict.

In December, 1882, Louisa Jane Taylor was tried at the Central Criminal Court, convicted, and subsequently executed, for the murder of Mrs. Tregelles by means of sugar of lead (lead acetate). The case is known as the Plumstead murder case. The following is a summary of the evidence offered at the trial:

Mr. and Mrs. Tregelles were an aged couple living in a small cottage at Plumstead in Kent. They were in humble circumstances, the husband having a pension of £42 a year. They were married in 1879, three or four years before the murder of Mrs. Tregelles. The husband at the time of his wife's death was eighty-six years of age, and she herself was eighty or eighty-one years old. Mrs. Tregelles was a fine hale woman with good white teeth, and she was accustomed to walk out unattended.

In March, 1882, Louisa Jane Taylor, a woman of nearly forty years of age, recently become a widow, called on the couple, her husband having at one time been a fellow official with Mr. Tregelles at the Woolwich Dockyard. On or about July 28th, 1882, Taylor went to reside with the Tregelles couple,

having obtained the permission of the husband to do so. They lived on the first floor, in two rooms, front and back, the rest of the house being occupied by the landlady, Mrs. Ellis and her husband. The back room was used as a bedroom by Taylor and Mrs. Tregelles, who occupied one bed; and the old man slept apart in the front room. Subsequently the rooms were changed, and the prisoner and Mrs. Tregelles occupied the front room. On August 10th, *i.e.* about twelve days after the woman Taylor went to reside with the couple, she took the old woman out for a walk. On their return Mrs. Tregelles was observed to have her eye cut; and this the prisoner attributed to her having been wantonly tripped up by a boy. She did not appear to suffer in other respects.

During this week, the second week in August, but the precise date is unknown, Taylor purchased an ounce of sugar of lead at the shop of Mr. Smith, a surgeon at Plumstead. She was served by Mrs. Smith, who placed a "Poison" label on the packet, and strongly cautioned Taylor as to its dangerous character. The sugar of lead was stated by Taylor to be required by her for a leucorrhœal discharge. Shortly after the alleged fall, as the evidence showed perhaps on August 11th, and the same week as the purchase of sugar of lead was made, the old lady's illness commenced. She was sick, was very poorly, had some diarrhœa and pains in the back. Next day the 12th she was confined to bed. As her illness persisted Mr. Smith was called in on August 23rd. The condition of Mrs. Tregelles was then as follows:—She was lying in bed, in a very low, weak condition. She complained of sickness, and Mr. Smith was informed that she vomited. There was some tenderness over the epigastrium and abdomen, and she had a colicky pain about the navel. There was sallowness of the skin and the teeth were dark. It will be remembered that up to July Mrs. Tregelles teeth were unusually white for a woman of her age. The tongue was dark brown, especially towards the root. The bowels were confined. She seemed very ill, and nothing could be retained in the stomach. No suspicion of the administration of poison was entertained; and her illness was attributed to ague and old age. Alkalies, ammonia, and occasionally small doses of hydrocyanic acid were given, and then quinine. Her condition improved, the sickness abating; and Mr. Smith ceased

to attend on September 6th. Though the prisoner who acted as nurse was frequently requested to save the vomits for Mr. Smith's inspection, she never did so, and invariably made some excuse for her neglect. The teeth it was observed became much darker, during Mr. Smith's attendance. The medicines he prescribed were mixtures, and occasionally a pill containing grey powder. No medicine in the form of powder was ordered; and this was an important feature in the case, as will be subsequently perceived, the prisoner having, as the evidence showed, beyond doubt administered a powder nightly to the deceased woman.

Three days later, viz. on September 9th, Mr. Smith was again called in by the husband, and on this occasion he attended Mrs. Tregelles one week, till September 16th. The sickness had returned. He again prescribed alkalies and hydrocyanic acid with a pill of grey powder at night. As she did not take his remedies he ceased his attendance on September 16th. She was described as being then low and feeble. Her teeth were much blackened—strikingly so.

On October 1st the old woman had what appeared to be an epileptic seizure whilst on the night-stool. At this time there had been no action of the bowels for the eight days preceding. There were rigidity of the limbs, some convulsion, the eyes fixed and staring, and twitching of the fingers. Later on in the course of the case the deceased became hemiplegic on the right side. On the 2nd October sickness returned after partaking of tinned lobster. At this time and throughout the illness the urine was very red, unusually so, according to all the witnesses who saw it: and stress was, as I believe unduly, laid upon this symptom by counsel, as being pathognomonic of poisoning by sugar of lead.

Four days later, on October 6th, suspicions of foul play were aroused. The prisoner had tried to induce the old man to desert his wife and live with her. One of the persons about the deceased suggested, on that day, that Mrs. Tregelles was being poisoned by sugar of lead; and this allegation being brought under the notice of Mr. Smith, the circumstance of the prisoner having on three occasions purchased sugar of lead at his own shop, came to light; and three important facts were subsequently established:—(1) That Mrs. Tregelles alleged that the prisoner

administered to her a white powder every night. (2) That the prisoner had stated to one of her own relatives that she had, by Mr. Smith's orders, to administer powders to the old woman. It will be remembered that Mr. Smith at no time ordered powders. (3) That an intelligent girl had seen the prisoner put a milky-looking fluid, not milk (? sugar of lead in tap-water), into food prepared for Mrs. Tregelles.

When Mr. Smith saw the deceased on October 6th she was very feeble, with loss of power and dropping and trembling of the hands. There were nausea and vomiting, with pain at the epigastrium and in the abdomen. Her condition was much worse than when he previously saw her three weeks earlier. The teeth were blackened, and this condition was most marked on the front teeth. The gums were now for the first time carefully examined, and the punctated blue gum-line of lead-poisoning detected. The line was very marked and characteristic, and was most developed on those teeth where tartar was most abundantly deposited. The conjunctivæ were of a dull yellow hue. Mr. Smith at once diagnosed lead-poisoning, of which he had seen several cases. The care of the patient was entrusted to Mrs. Ellis, the landlady of the house. No sooner was the old lady relieved of the presence of the prisoner than her condition began to temporarily ameliorate, and the sickness left her. On the 9th October the Divisional Surgeon of Police, Dr. Sharpe, saw her in consultation with Mr. Smith, and confirmed this gentleman's diagnosis. He had no doubt of the presence of the lead-line, that her state was due to the ingestion of lead, and that the paralysis of the hands was owing to the same cause. During the first week, under the influence of potassium iodide, the patient rallied, but on October 13th she became hemiplegic on the right side, and died on the 23rd. No analysis of the urine was made during life.

It may be here remarked that the deceased had frequently complained of the peculiar taste of the medicine given her by the accused; and also that Taylor purchased sugar of lead on three occasions during a period of about six weeks—two or two and a half ounces on the whole. None of this was found after her arrest, nor, indeed, any article containing lead acetate.

A post-mortem examination was made by Mr. Smith and Dr. Sharpe on the 24th—the day after death—in the presence

of Mr. Atkins, a medical man who watched the case on behalf of the accused. The skin was of a sallow icteric hue, more especially upon the upper part and front of the trunk. There was a distinct blue lead-line on the gums. The only abnormality in the brain was an atheromatous condition of the arteries at its base. The lungs exhibited the usual congestion of decubitus. The pulmonary artery was thickened. The heart was small but healthy. The liver was healthy, but one lobe was unusually small. The spleen was healthy, and weighed three ounces. The cortex of the kidneys was wasted, but not more than is usual at such an advanced age. Their aggregate weight (seven ounces and a quarter) was normal. The bladder was healthy in appearance, and contained urine. Unfortunately this was not preserved for analysis.

It was in the alimentary canal that the appearances were most marked. The mucous membrane of the stomach exhibited dark brown, almost black, streaks all over its surface, but these were most marked over the pyloric half. But the small and large intestines exhibited on their mucous surfaces many slatey, almost black patches, such as are, according to my experience, not infrequently met with in chronic lead-poisoning. The large intestine is also said to have been contracted in this case.

The condition of the viscera when they reached me for analysis precluded a satisfactory microscopical examination of the solid organs. The portions submitted for analysis were a portion of the brain, lung, liver, and spleen, two kidneys, and a portion of the ileum, cæcum, and colon.

Portions of these viscera were very carefully dried and incinerated at a low temperature in a muffle, and care was taken to extract the whole of the lead from the coal by the successive use of dilute nitric acid, and solution of ammonium acetate. The coal was in each case again incinerated and re-extracted with the above-named solvents. The metal was precipitated as sulphide by sulphuretted hydrogen, dried and weighed. The lead sulphide was then converted into sulphate by means of nitric and sulphuric acids and weighed. The quantities of lead calculated from the sulphide and sulphate respectively were accordant. The lead sulphate was in each instance tested for.

copper, but mere traces only of this metal were found in the precipitates from the liver and brain alone.

By subsequent operations the lead sulphate was converted into the corresponding chromate and iodide, and fine crystals of the latter were obtained for production at the trial.

In this way the following quantities of lead were actually obtained :

2½ ounces of stomach yielded . . .	0·22 grain.
Slatey portions of intestines yielded .	trace.
Undarkened „ „ .	no lead.
8 ounces of brain „ .	0·05 grain.
16 „ liver „ .	0·26 „
3 „ spleen „ .	trace.
3 „ kidneys „ .	0·01 grain.
2 „ lung „ .	trace.

Calculated on the whole viscera these quantities are :

Stomach	0·44 grain.
Slatey portions of intestines	trace.
Brain	0·42 grain.
Liver	0·56 „
Spleen	trace.
Kidneys	0·02 grain.
Lungs	trace.

Thus the aggregate amount of lead in the stomach, brain, liver, and kidneys, was nearly one grain and a half.

After the conviction of the prisoner the portions of viscera kept in reserve were submitted to a check analysis by a different method, the organic matters being destroyed by a mixture of nitric and sulphuric acids, as recommended by Gautier. Assuming that these portions contained the same proportions of lead as those previously submitted to analysis, 0·28 grain of lead should have been found—0·25 grain was the amount actually obtained.

I have already referred to the somewhat characteristic appearance of the mucous membrane of the intestines—dark slatey patches with intervening less highly-coloured portions. A somewhat similar condition was observed in the stomach. This condition I have frequently observed in the intestines in lead-poisoning. My attention was first directed to it by Dr. Fagge. Invariably in such cases I have found a much higher

percentage of lead in the slatey than in the uncoloured portions of intestine.

It will be noted that the quantity of lead found in the stomach was relatively large, and it was this fact which enabled me to state with some confidence that doubtless the poison had been taken through the mouth, probably in considerable quantity, and at no distant date. The case presented some grave difficulties, but the above circumstances, when taken into consideration with the history of the case, nauseating taste, sickness, colic, constipation, epileptiform seizure, loss of power in the hands, a marked blue gum-line, and the peculiar darkening of the teeth, probably due to the action of the buccal secretions upon the lead salt, afford strong evidence that the death of Mrs. Tregelles was *accelerated* by the ingestion of lead. No other person in the house showed any symptoms of lead-poisoning, and the water-supply was proved to be free from lead.

The moral and circumstantial evidence against the prisoner was unusually strong, and secured her conviction; and I have been informed that before her execution the prisoner admitted the justice of her sentence.

The case is perhaps an unique one in English medical jurisprudence, and hence is worthy of being recorded.

It has been recently stated by M. Armand Gautier that ordinary persons habitually take lead to the extent of at least $\frac{1}{130}$ th of a grain daily in their food. My own extensive experience leads me to the conclusion that it is exceptional to find a trace of lead in the body except in cases of lead-poisoning, but that, on the other hand, it is equally exceptional not to find traces of copper in the human body after death.

ON

THE VITREOUS BODY IN ITS RELATION
TO VARIOUS DISEASES OF THE EYE.

By W. A. BRAILEY, M.D.

THE condition of the vitreous body in different diseases, more especially in their earlier stages, appears to be a subject worthy of more consideration than has hitherto been bestowed upon it.

It is difficult, however, here, as in the case of other diseases of the eye, to obtain exact knowledge unless the morbid changes have been so grave as to warrant enucleation.

But information thus acquired requires to be supplemented by a careful watching in the post-mortem room for the rarely-occurring cases where patients, the subjects of eye affections, have died of other maladies, and by examinations, both macroscopical and microscopical, of the vitreous where it has happened to escape during operations for the relief of glaucoma, cataract, and other morbid conditions.

The following observations concern the consistency, size, general appearances, and microscopical characters of the vitreous body as found in excised eyes.

In Glaucoma an examination of many cases of varying duration leads me decidedly to the conclusion that the vitreous is unduly firm and consistent in the earlier stages of

this disease. But, when incised, its bulk becomes slowly diminished owing to the draining away of a considerable quantity of clear fluid, which thus appears to have been enclosed within its substance more closely than in the normal condition of health.

When the disease is of considerable standing, *e.g.* one or more years, the posterior part of the vitreous may be rather more fluid than normal, though it is by no means invariably so. In some cases I have found it of at least normal consistency when increase of tension has lasted for some years. When, however, the globe has yielded to the distending force, the vitreous is always more fluid than normal, corresponding, in fact, pretty much in appearances with the vitreous of myopia with posterior staphyloma.

It is a striking characteristic of glaucoma, as compared with most other diseases, that the vitreous is rarely found shrunken and by consequence detached.¹ Even if there be considerable fluidity at and near the posterior pole we still find a little vitreous adhering to the retina of this region, while its anterior part still retains, as in most other conditions, its attachments in front of the ora serrata, not presenting that well-defined posterior surface which would be seen were the vitreous shrunken and detached from the retina.

I must, however, remark that this rule does not apply to the cases where the glaucoma is secondary to tumours or to severe iritis, whether traumatic or not, for in these cases the vitreous shrinks, as will be afterwards described.

From what has been said it might suggest itself that the enlargement of the vitreous body is the cause of the increased tension in primary glaucoma. In many cases we must admit this to be the fact, for there can be no doubt but that the vitreous is the agent by which the optic disc in one direction, and the lens in the other, are pressed upon. The same is the case with regard to the scleral staphylomata occasionally met with, though there are cases in which the retina at these spots has become so thickened by the accumulation of fluid in its substance, between Müller's fibres (œdema or cystic disease of

¹ Pagenstecher and Genth, in plate ix of their 'Atlas,' figure an eye with chronic glaucoma in which the vitreous is transformed into a membrane, but there are no notes either of the history or clinical appearances.

the retina¹), as to nearly occupy the extra space gained by the staphylomatous bulging.

On the other hand, we know that the tension may become increased in eyes with shrunken vitreous, as, for example, with choroidal tumours, and iritis with excluded pupil, &c. In these cases, however, the tension is clearly secondary to the other morbid conditions, and is due to the accumulation of fluid in the subretinal space behind the detached vitreous, or even in the posterior chamber. There are also cases of glaucoma in which the tension is associated with, and even due to, large hæmorrhages in the subretinal space. Still it would require a very large experience, both clinical and pathological, to justify one in saying that a primary glaucoma never occurs in eyes with unshrunken vitreous.

From the physical conditions of the case it is difficult, if not impossible, to prove by measurements that the vitreous is actually enlarged in primary glaucoma. But, whatever may be the fact in the primary glaucoma of senile eyes, it is obvious that this is really so in those cases where the vitreous still fills its cavity notwithstanding, as is almost invariably the case in the glaucoma of young persons, that the eye has become considerably increased in size from distension of its tunica from within.

The vitreous appears transparent in the majority of cases, but in the recent, and especially in the acute ones, it contains ill-defined, opaque streaks and spots. These are more frequent in its peripheral parts and not infrequently radiate from the optic disc. They are interesting when taken in association not only with the condition of the optic disc, which is usually found swollen in eyes excised on account of recent acute glaucoma, but also with the connective-tissue which so commonly forms a lining to the glaucoma cup. The above-described condition of the vitreous, which is presumably inflammatory, may be associated with the constantly occurring glaucomatous inflammation of the optic nerve and ciliary body.²

¹ Iwanoff, 'A. F. O.,' xv, pt. 2; also Nettleship, 'R. L. O. H. Rep.' vol. vii, pt. 3.

² Brailey and Edmunds "On the Optic Nerve, Ciliary Body, and Iris, in Increased Tension," 'R. L. O. H. Rep.,' vol. x, pt. 1.

Hæmorrhages into the vitreous are, as is well known, not uncommon in glaucoma. They may be from the ciliary body, but are far more common from the retina, and even from the papilla itself. Sometimes the whole vitreous is tinged yellow as if from blood-staining. The hæmorrhages are usually small and multiple. They present the same characters when they have preceded the outbreak of the tension (*G. hæmorrhagicum*).

Microscopical examination shows marked changes of the vitreous, whether the glaucoma be acute and recent or of longer duration and slower progress. There is in every case, even though the vitreous be unduly fluid, an increase in its corpuscular elements. Such may be either round or slightly angular in shape, with not very well-defined outline and one or two well-defined nuclei, and in size varying from $\cdot 013$ to $\cdot 017$ millimètres in diameter. Or they are tailed, being stellate or more frequently drawn out at two ends so as to approach or attain to the condition of well-defined spindle cells, each with a central enlargement containing one or two nuclei. The typical spindle cells measure $0\cdot 056$ millimètres in length by $0\cdot 01$ millimètres at their thickest part. The round and spindle forms are usually intermixed, though more commonly either the one form or the other is in great excess. In some cases I have found cells corresponding in appearance to the signet-ring-like cells of the normal vitreous. As a rule such cannot be found, probably simply on account of their rarity. In one case, however, they attained a number considerably beyond the healthy average.¹

We may suppose that there is in glaucoma an increased serous exudation into the vitreous body together with an increase of its cell elements, the whole process being of an inflammatory nature.² Still the inflammation here found is entirely different in degree and even in kind from that of suppurative and even of non-suppurative traumatic hyalitis. In glaucoma the vitreous does not render Müller's fluid opaque, whereas in the other morbid conditions just mentioned, the opacity both of the vitreous and of the fluid in which it has been preserved is extreme.

As to the causal relation between the inflammation of the

¹ See in this connection, Iwanoff, in '*A. F. O.*,' xi, pt. 1.

² See Stilling, in '*A. F. O.*,' xiv, pt. 3.

optic nerve and that of the vitreous it would appear that the hyalitis of glaucoma is secondary in point of time to the glaucomatous inflammation of optic nerve and ciliary body, and that the one inflammation has given rise to the other by direct extension. For many cases are observed clinically in which a swollen disc with clear, and, therefore, presumably normal vitreous, precedes the increased tension and invisible fundus of glaucoma. The same is probably the sequence in glaucoma secondary to embolism of the arteria centralis or plugging of the central vein of the optic nerve. Still we must remember that the obscuration of the fundus in glaucoma is attributed to other causes than opacity of the vitreous, and that the great majority of cases of optic neuritis are not succeeded by glaucoma or any noticeable change in the vitreous. This is especially the case with neuritis or rather papillitis from intra-cranial causes, though it may be said on the other hand that the swelling of the papilla in such cases exceeds vastly the degree of inflammation of the nerve trunk, and, therefore, it may be more of the nature of an œdema or inflammatory œdema than an active inflammatory change.

In this connection may be noted the constancy of swollen disc in association with wounds of the anterior parts of the eye. I used to account for the papillitis which accompanies ulcers or wounds of the cornea by supposing that the fibrous tissue of the sclerotic had been the means of transmitting the inflammatory change. It was first suggested to me by Mr. Nettleship that the vitreous might form the track along which the inflammation travelled. This, it now appears to me, may well be so.

In the case of injuries of the cornea with or without accompanying iritis we find that the inflammation of the vitreous body, though always clearly distinguishable from that accompanying glaucoma, differs somewhat in its characters in accordance with the extent of the injury and its duration.

From slight injuries with no perceptible change in the vitreous we pass to more severe ones in which this body is found rather more consistent than normal, clear or only slightly hazy, and separating readily from its posterior attachments on slight manipulation. When incised it retains

its consistency for some time, there being no such considerable draining away of fluid as we see in glaucoma.

In traumatic cases where the vitreous itself is not directly involved, microscopic examinations undertaken within a couple of days of the injury reveal nothing abnormal. Examinations of cases of slightly longer duration show that the vitreous, unless entirely unaffected, contains an excess of cell elements, mostly round, $\cdot 013$ to $\cdot 017$ in diameter, and each having a single or double nucleus. Tailed cells are rare. In addition to the above there are found other cells evidently corresponding to the signet-ring-like cells of the normal vitreous. These are round, transparent, vesicular-looking bodies of sharp outline, each presenting on its surface a single well-defined nucleus. The cells nearly always lie in such a position that the nucleus, which also looks vesicular, seems to be on one side, the whole having a signet-ring-like appearance. Such cells are so rare in the normal eye as to be found with difficulty by the microscope. But in the comparatively slight non-suppurative hyalitis, which so commonly complicates wounds of the cornea or iris and cornea, such cells are very numerous, existing in some cases in the proportion of one to every three of the smaller round cells just described. Their frequency contrasts strongly with the condition in glaucoma where such cells are usually extremely rare, and but singly nucleated. Another characteristic point lies in their shape, the nuclei usually being larger and the cells smaller than in the normal condition, so that the relative size of nucleus and cell body is very considerably altered. Or the nucleus is not uncommonly double so that we have the appearance of a signet-ring with a double stone.

In these different microscopic characters, the frequency of the cells both relative and absolute, the relative size of nucleus and cell and the frequently double nucleus, the non-suppurative hyalitis of injuries differs from that of glaucoma.

The vitreous may remain in this condition for long after the injury, even for many years, but if the inflammation have been more severe, so as to involve the uveal tract more extensively, the tendency is for the vitreous to shrink.

In such cases this body forms a transparent or translucent globe adhering closely to its attachments in front of the ora

serrata. Its posterior clear and well-defined surface is obviously bounded by the hyaloid, and is separated by a considerable fluid-containing space from the retina which remains still *in situ*.

Microscopically we find the spindle-cells to be abundantly intermixed with the small round form, and the signet-ring-like cells to be comparatively very rare.

In injuries which have given rise to sympathetic inflammations of the opposite uveal tract the condition of the vitreous is much the same as that described above as occurring in severe traumatic inflammations, with which they are evidently closely allied, if not identical. But many cells which have the appearance of multiplying themselves by budding are also conspicuous in the vitreous. Such are rounded in their general outline, but send off one or more short processes terminating in rounded ends. Occasionally an end is considerably broader than its connecting stalk, so that the terminal part looks as if it were about to be detached as a separate cell. These ramifying cells are nucleated and have their outline moderately well defined. The highly organisable products of such inflammations may, by their mutual adhesion and contraction, draw in not only the retina, but in some cases the choroid also with its adherent sclerotic, and thus produce shrinking of the eyeball.

Suppurative hyalitis stands commonly as to the rapidity of its development in marked contrast with the slower processes of non-suppurative inflammation. In its earlier stages the vitreous is unduly consistent and slightly turbid. It separates readily from the posterior part of the globe except in the immediate neighbourhood of the swollen disc. Fine opaque streaks corresponding to the position of the retinal blood-vessels remain on the vitreous when separated from the retina. A thin, greyish film may clothe the whole or part of the ciliary processes and also the adjacent lens margin. The development of such changes has already been described by the author.¹ They result in the shrinking of the vitreous, and sometimes in its almost total absorption. The retina may remain *in situ*, but is usually detached. If the choroid have

¹ Hebb and Brailey "On the Phenomena of Suppurative Hyalitis," R. L. O. H. Rep., vol. x, pt. 2.

participated in or originated the suppurative changes (suppurative panophthalmitis) it retains its connections with the retina rather more closely, and through it with the shrinking vitreous, and thus we have shrinking of the whole eyeball. The nîsus of development is towards producing an immense number of imperfectly-developed quickly-degenerating cells, which rapidly come to occupy the entire vitreous.

Experiment shows the slow reaction of the vitreous to inflammatory influences, even when it is directly involved in the injury. When in the rabbit the vitreous has been partially evacuated, I have found, on opening the eye several hours later, the remaining portion to contain no excess of cell-elements, though it is abundantly infiltrated with albuminous matter coagulable by Müller's fluid. The same has been the case in human eyes excised twenty-four hours after iridectomy or sclerotomy. But at the end of two days the vitreous begins to present more decided microscopical changes, as described above.

Judging from the advance of the lens and the occasional occurrence of equatorial staphylomata with the increased tension of early suppurative hyalitis, we may infer that the vitreous is actually enlarged. Moreover, we can see the ligamentum pectinatum to be swollen in such cases when infiltrated with pus cells, and the sclera and episcleral tissue to be thickened by albuminous infiltration. As a rule there is no evidence of any such swelling of the vitreous in traumatic non-suppurative inflammations. It is, however, true that the anterior chamber is shallow in many of the severe traumatic inflammations which produce sympathetic disease, while as yet the tension is increased and the vitreous unshrunk. It may, therefore, be that the enlarged vitreous is the cause of the increased tension encountered in this condition, as it is the cause of the tension in suppurative panophthalmitis. The vitreous of suppurative, like that of severe traumatic non-suppurative, hyalitis renders Müller's fluid turbid, and in this respect stands in marked contrast with the hyalitis of glaucoma.

There are other conditions in which the vitreous is found constantly changed. In intra-ocular tumours it shrinks even more rapidly than the tumour grows, so that the retina is found detached unless the growth be in its very earliest

stages. This shrinking of the vitreous is observed long before any increase in the intra-ocular tension has taken place. It may result in its almost total disappearance, so that we might infer that the process is not an inflammatory one.

In myopia it is well-known that the vitreous is of an undue fluidity. This condition is probably connected with the increased size and thinned tunics of the globe. De Wecker¹ and other authorities describe an increase in the cell elements to which the opacities occasionally met with are due.

In the secondary glaucoma of young persons consecutive to old perforating ulcers with entanglement of iris, the changes, when advanced, are precisely similar to those of myopia, as the vitreous entirely occupies its chamber and its posterior part is fluid in association with the thinning of its tunics. It also contains an excess of cell elements.

But the vitreous, though enlarged in such cases of secondary glaucoma, is not necessarily of undue fluidity. I have satisfied myself by repeated observations that there are many cases of glaucoma secondary to perforated corneal ulcers in young persons in which the vitreous, though enlarged in exact association with the enlargement of the globe, is even of abnormal firmness. It would appear to be the subject of an excessive infiltration of serous fluid associated with increase of its cell elements. The changes would thus appear to be inflammatory and closely allied with those of primary glaucoma.

Possibly they are of a mixed nature, as we find by microscopical examination an excess of the signet-ring-like cells as well as of the other forms common after injuries.

The changes in iritis, whether of rheumatic or syphilitic origin, follow very closely those of severe wounds, the tendency being to the formation of spindle cells with rare signet cells and ultimate shrinking of the vitreous. The vitreous may often be found shrunken with or without detachment of the retina, where such an iritis has produced a glaucoma.

In sloughs or suppurations of the cornea the vitreous may present suppurative changes, or it may undergo the inflammation ordinarily accompanying the severer injuries of cornea and iris.

¹ Wecker and Jaeger's 'Atlas,' &c.

What is the practical importance of the hyalitis accompanying these different morbid conditions?

If in most cases of primary glaucoma the vitreous is, as I believe, the distending agent, it is, though only secondary in time to the inflammation of the optic nerve and ciliary body, still of the greatest significance, for most of the symptoms of glaucoma are immediately referable to the intra-ocular pressure. That the tension may become increased in eyes with detached retina or shrunken vitreous, whether from tumours or other causes, does not prove much, unless it can be established that the glaucoma is a primary one and not merely secondary to the cause producing these conditions.

It may be reasonably supposed that the glaucoma frequently occurring after traumatic dislocation of the lens is dependent upon a change in the vitreous rather than upon any pressure by the displaced lens upon the ciliary body or iris, for in many such cases the lens is seen to be suspended in the vitreous chamber from its upper attachments only.

I cannot as yet say that an increased consistency of the vitreous is absolutely necessary for the establishment of a primary glaucoma.

When glaucoma breaks out in a myopic eye the presumption, in the absence of post-mortem evidence of recent cases to the contrary, is that the vitreous is more fluid than normal, just as it is in ordinary myopia.

In the glaucoma secondary to excluded pupil, the vitreous is frequently found shrunken and detached from the retina, appearing to follow in its condition the iritis to which the glaucoma is secondary.

In the case of choroidal tumour, as before explained, it invariably shrinks rapidly.

In traumatism the changes in the vitreous produce fewer evils. If the wounds are severe they may be followed by increased tension, which may or may not be due to swelling of the vitreous. Certainly the vitreous is often turbid after injuries without any accompanying manifestation of increased tension; and an eye may subsequently become hard notwithstanding that the injury has occasioned loss of vitreous. I should be inclined to say that a traumatic hyalitis might be attended with swelling of substance, and consequently in-

creased tension if the inflammation partakes of the serous or suppurative character, but not if the inflammation is of the character above described as usually following wounds.

If the ordinary traumatic hyalitis is extremely severe, as in the cases exciting sympathetic disease, the shrinking vitreous may by its union with the retina, and through this with the inflamed choroid and the sclerotic, cause shrinking of the globe, but if the wounds and accompanying hyalitis be not of so severe a character, the shrinking vitreous may leave the retina and choroid *in situ*. But in either case the condition of the vitreous does not influence the vision much, as this is rather dependent on the degree of implication of the retina and choroid. And precisely the same may be said with regard to suppurative hyalitis or panophthalmitis.

In choroidal tumours the detachment of the retina is caused rather by an accumulation of subretinal fluid than by the shrinking of the vitreous.

In myopia the fluidity of the vitreous appears to be a matter of considerable importance, as it is usually supposed that it is the cause of the retinal detachments so common in high degrees of this condition.

It would appear that all abnormal conditions of the vitreous, excepting possibly intraocular tumours, are accompanied by an increase of its cell elements.¹

Stilling has suggested² that an accumulation of fluid in the hyaloid canal may be the cause of a glaucoma. As before said, I myself have found no evidence of any such accumulation in this position, and it appears difficult to reconcile any such supposition with the occurrence of local staphylomata whether with or without corresponding areas of oedematous retina (see page 486-7). It rather looks as if the change were within the substance of the vitreous itself and as if it might even be localised in a particular part of this body.

¹ See Wecker and Jaeger's 'Atlas, "Myopia, with Sclero-choroiditis posterior"; also Poncet's 'Pathological Atlas,' "Synchysis."

² "Zür Theorie des Glaucoms," 'A. F. O.,' xiv, pt. 3.

STATISTICAL SUMMARY

OF

PATIENTS TREATED IN GUY'S HOSPITAL DURING THE
PAST FOUR YEARS (1879—1882).

By J. C. STEELE, M.D.

	1879	1880	1881	1882
Patients in hospital 1st Jan. each year .	538	562	433	440
Admitted annually	5189	4627	4490	4710
Discharged as cured	1733	1820	1737	1759
Relieved	2469	2060	1957	2242
Unrelieved	418	399	329	258
Died	545	477	465	451
Remaining 31st Dec. each year	562	433	435	440
Average number resident daily	566	506	456	453
Mean residence of each in days	39·2	39·5	36·7	34·4
Rate of mortality per cent.	10·55	10·20	10·36	9·57
MEDICAL WARDS.				
Patients in hospital 1st Jan. each year .	225	238	161	156
Admitted in the course of the year	2087	1738	1593	1656
Discharged as cured	379	375	406	498
Relieved	1108	931	771	772
Unrelieved	211	196	144	114
Died	376	313	282	278
Remaining 31st Dec. each year	238	161	151	150
Average number resident daily	236	192	156	153
Mean residence of each in days	39·6	40·7	36·4	34·0
Rate of mortality per cent.	18·12	17·30	17·59	16·73
SURGICAL WARDS.				
Patients in hospital 1st Jan. each year .	313	324	272	284
Admitted each year	3102	2889	2897	3054
Discharged as cured	1354	1445	1331	1261
Relieved	1361	1129	1186	1470
Unrelieved	207	203	185	144
Died	169	164	183	173
Remaining 31st Dec. each year	324	272	284	290
Average number resident daily	330	314	300	300
Mean residence of each in days	38·9	39·1	37·2	34·7
Rate of mortality per cent.	5·46	5·55	6·34	5·82

Summary distinguishing the Sexes with the relative Mortality.

	MEDICAL WARDS.						SURGICAL WARDS.					
	Admitted.		Discharged.		Died.		Admitted.		Discharged.		Died.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
1879	1068	1019	836	862	226	150	2041	1061	1927	995	121	48
1880	1001	737	842	660	193	120	1900	989	1810	967	113	51
1881	953	640	772	549	181	101	1865	1032	1735	967	113	70
1882	975	681	791	593	183	95	1972	1082	1858	1017	115	58

Retrospective Summary of In- and Out-Patients who have received Relief at the Hospital during the past ten years.

	1873.	1874.	1875.	1876.	1877.	1878.	1879.	1880.	1881.	1882.
IN-PATIENTS.										
Under treatment during the year . . .	5,571	5,776	5,854	5,722	5,544	5,710	5,727	5,189	4,923	5,150
Discharged cured . . .	1,400	1,354	854	1,511	1,550	1,551	1,723	1,820	1,737	1,759
Relieved . . .	2,749	2,925	3,452	2,664	2,493	2,695	2,469	2,060	1,957	2,242
Unrelieved . . .	341	334	419	435	460	371	418	399	329	258
Died . . .	524	594	560	593	498	555	545	477	465	451
Rate of mortality per cent. . .	10.44	11.40	10.60	11.39	9.95	10.72	10.55	10.24	10.36	9.57
Mean residence of each in days . . .	39.8	38.6	37.3	39.5	39.5	37.9	39.2	39.5	36.7	34.4
Number of accidents . . .	885	852	928	871	803	824	935	869	994	997
Deaths from accidents . . .	85	102	95	83	81	76	77	73	78	72
OUT-PATIENTS.										
Surgical cases . . .	3,681	3,801	3,313	3,505	3,988	3,919	3,534	3,928	3,195	3,661
Medical cases . . .	3,126	2,919	2,987	3,265	3,041	2,838	2,884	2,773	2,843	2,703
Diseases of the eyes . . .	3,356	3,083	2,717	3,252	3,028	3,059	2,552	2,654	2,286	2,502
Diseases peculiar to women . . .	1,719	1,644	1,745	1,902	2,114	1,963	1,416	1,540	1,277	1,400
Diseases of the skin . . .	994	989	1,170	985	1,000	1,031	963	947	923	1,013
Diseases of the ear . . .	1,332	1,089	990	1,251	1,318	1,189	1,060	1,072	939	1,107
Casual or minor medical cases . . .	8,532	7,042	7,143	7,013	7,535	8,107	7,863	9,056	11,810	12,854
Casual or minor surgical cases . . .	41,670	43,573	41,363	38,649	35,247	21,934	17,781	20,818	23,670	26,046
Tooth extractions . . .	2,204	3,758	2,278	2,783	2,801	3,261	3,695	2,850	16,320	1,346
Minor accidents . . .	10,770	14,746	9,764	11,005	11,449	11,353	11,020	12,173	13,170	12,909
Women confined at their homes . . .	2,213	2,449	2,334	2,449	2,540	2,552	2,613	2,593	2,483	2,504
Deaths after confinement . . .	16	17	9	13	9	5	3	8	6	7

L I S T
OF
GENTLEMEN EDUCATED AT GUY'S HOSPITAL
WHO HAVE PASSED THE
EXAMINATIONS OF THE SEVERAL UNIVERSITIES, COLLEGES,
&c., &c.,
IN THE YEAR 1881.

University of Oxford.

First Examination for the Degree of Bachelor of Medicine.

F. B. W. Phillips. | W. Growse.

University of Cambridge.

Final Examination for the Degree of Bachelor of Medicine.

J. E. Viney. | R. A. Birdwood.
W. H. C. Newnham.

Second Examination for the Degree of Bachelor of Medicine.

R. L. Knaggs. | J. H. H. Manley.

First Examination for the Degree of Bachelor of Medicine.

W. A. Bell.

University of London.

Examination for the Degree of Doctor of Medicine.

J. W. Meek.

Logic and Psychology only.

W. H. Lamb. | R. S. Wainewright.

Final Examination for the Degree of Bachelor of Medicine.

First Division.

W. A. Lane.

Obtained the Gold Medal in Medicine, and Honours in Forensic Medicine:

500 Gentlemen admitted to Degrees, &c., in the year 1881.

B. N. Rake.

*Obtained First-Class Honours in Medicine, Honours in Obstetric Medicine,
and Honours in Forensic Medicine.*

R. Bredin.

Obtained First-Class Honours in Medicine and Honours in Obstetric Medicine.

H. T. Bassett.

Second Division.

G. R. Marsh.

Obtained Honours in Forensic Medicine.

John Smith.

Intermediate Examination in Medicine.

First Division.

W. Hind.

*Obtained the Exhibition and Gold Medal in Organic Chemistry, and First-Class
Honours in Physiology and Histology.*

J. H. Targett.

*Obtained Honours in Anatomy, in Physiology and Histology, and in
Organic Chemistry.*

A. Martin.

Obtained Honours in Anatomy.

L. A. Dunn.

Second Division.

E. J. Wenyon.

*Obtained the Gold Medal in Anatomy, and Honours in Physiology and
Histology.*

M. Carnelley.

R. Cuff.

C. H. L. Meyer.

M. Parry-Jones.

J. H. Champ.

W. W. Floyer.

M. O'Kane.

J. Maughan.

Excluding Physiology.

C. Gross.

Physiology only.

R. Parry.

Preliminary Scientific (M.B.) Examination.

First Division.

W. L. Braddon.

Obtained Honours in Botany.

C. Caldecott.

Obtained Honours in Inorganic Chemistry.

J. Chadwick.
E. W. Goodall.
E. Goodall.
J. G. Harsant.
H. Rockley.

F. B. W. Phillips.
A. E. Price.
R. M. H. Randell.
S. G. Rawson.
G. Rowell.

J. W. Washbourn.

Second Division.

S. R. Alexander.
E. H. Armitage.
H. H. Du Boulay.

E. P. Mourilyan.
J. H. Sellick.
M. G. Younge-Bateman.

Final Examination for the Degree of Bachelor of Surgery.

First Division.

W. A. Lane.

Obtained Honours in Surgery.

University of Durham.

Final Examination for the Degree of Doctor of Medicine.

W. J. Tyson.

Final Examination for the Degree of Bachelor of Medicine.

H. H. Austin.

H. A. Clowes.

Final Examination for the Degree of Master of Surgery.

H. A. Clowes.

First Examination for the Degree of Bachelor of Medicine.

J. V. Salvage.
H. H. Austin.
A. T. F. Brown.
C. Y. Shuter.

W. E. Paley.
J. I. Parsons.
H. P. Keatinge.
G. H. Rodman.

University of Edinburgh.

Final Examination for the Degree of Bachelor of Medicine.

G. C. S. Perkins.

University of Aberdeen.

Examination for the Degree of Doctor of Medicine.

A. H. Burton.

E. Field.

D. D. Malpas.

British Medical Service.

London Examination in February.

T. C. Nugent, 1620 marks.

G. S. Lewis, 1610 marks.

R. E. R. Morse, 1600 marks.

Nelley Examination in August.

H. O. Stuart, 2125 marks.

C. W. S. Magrath, 1830 marks.

F. T. Wilkinson, 1370 marks.

502 *Gentlemen admitted to Practice, &c., in the year 1881.*

Royal College of Physicians.

Final Examination for the Licence.

S. R. H. Mathews.	H. A. Clowes.	H. Hawksworth.
O. B. Shelswell.	E. A. Starling.	P. Warner.
J. G. Barns.	F. Hitch.	F. W. Pilkington.
J. S. Crook.		

First Examination for the Licence.

March.		
A. J. Dalton.	C. F. Wakefield.	F. W. Pilkington.
E. S. Tresidder.	J. O. Littlewood.	W. R. Etches.
E. A. Starling.	R. H. Perks.	J. G. Barns.
July.		
W. E. Andland.	H. C. Dixon.	R. J. Kerby.
B. P. Bartlett.	S. B. A. Edsall.	A. Scott.
T. W. L. Beales.	A. P. H. Griffiths.	P. Warner.
J. S. Crook.	C. J. Harper.	J. H. H. Williams.
T. M. Day.	H. Hawksworth.	W. Wilson.
October.		
A. W. Clark.	E. Roberts.	D. C. Trott.
G. A. Johnson.	J. M. Owen.	A. H. Tubby.
E. E. Masters.	H. I. Tresidder.	W. Watson.
M. A. Muirhead.		
December.		
A. Bolton.	G. H. Graham.	P. Pigott.
A. J. Carter.	F. E. Hubbard.	G. W. B. Slader.
H. L. Cortis.	A. G. Minns.	A. S. Topham.

Royal College of Surgeons.

Final Examination for the Fellowship.

W. T. Crew.	C. J. Symonds, M.S.	J. C. Uhthoff, M.D.
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First Examination for the Fellowship.

S. Worthington.	H. W. Pigeon.	R. L. Knaggs.
W. Hind.	L. A. Dunn.	J. H. Targett.

Final Examination for the Membership.

January.		
C. H. Downes.	W. E. Fielden.	G. R. Marsh, M.B.
B. Scott.	A. Bolton.	E. A. Starling.
H. Hawksworth.	T. A. J. Shepherd.	C. Crossley.
G. S. Mahomed.	T. R. Atkinson.	W. H. Puddicombe.
A. S. Stokes.	S. T. Thomas.	J. M. Prendergast.
J. I. Palmer.	B. Studer.	
April.		
F. E. Row.	F. H. Shaw.	J. I. Boswell.
L. Stokes.	W. P. Morgan.	J. Rigby.
P. Warner.	T. B. Luscombe.	J. J. Udale.
H. R. Osborne.	O. J. Currie.	J. M. Owen.
L. Burroughs.	J. E. Anderton.	J. W. Woodruff.
F. N. Pedley.	G. F. Dixon.	O. B. Shelswell.

Gentlemen admitted to Practice, &c., in the year 1881. 503

July.

R. H. Perks.	Z. Prentice.	R. A. Milligan.
H. Blatherwick.	H. W. Phillips.	G. T. Woolley.
J. Dowson.	E. S. Dashwood.	A. T. Perkins.
E. N. Davies.	T. Unicume.	H. H. Austin.

November.

B. R. A. Taylor.	S. O. Stuart.	E. Elliott.
G. N. Pitt.	W. H. C. Strachan.	H. E. Archer.
L. E. Shaw, M.B.	W. C. Dendy.	P. H. Gardner.
E. L. Adeney, M.B.	G. P. Longman.	W. J. Coles.
W. H. Hart.	C. R. O. Garrard.	

First Examination for the Membership.

January.

E. D. Minter.	F. Pearse.	H. H. W. Button.
A. L. Paliologus.	J. F. G. Dill.	T. B. Winter.
E. Wakelam.	D. T. Key.	W. Howard.

April and May.

W. L. Blight.	H. J. Dring.	A. Linnell.
A. W. Clark.	E. W. Simmons.	A. C. Deare.
E. E. Masters.	A. G. Minns.	W. A. Aikin.
W. Watson.	W. Fowler.	F. Heatherley.
G. A. Johnson.	J. H. H. Manley.	A. J. Carter.
A. Martin.	W. H. Brenton.	H. R. Mead.
G. H. Rodman.	M. M. Adler.	A. E. Taylor.
F. B. W. Phillips.	C. D. Muspratt.	A. L. Scott.
H. E. Counsell.	C. W. Crealock.	W. L. W. Marshall.
A. E. Larking.	H. I. Tresidder.	G. E. Stewart.
C. S. Jago.	J. P. Martin.	E. H. Armitage.
A. H. Dodd.	H. Lamb.	J. C. Bates.

July.

H. J. Hillstead.	W. I. Watson.	C. E. Bean.
G. F. Hugill.	E. Sharpley.	L. J. Kidd.
F. W. H. Penfold.	H. E. Jones.	E. Linnell.
A. P. Rainbird.	H. Howard.	R. T. Westbrook.
C. S. Spong, B.Sc.	W. T. Hodge.	J. I. Parsons.
H. W. Whyte.	J. Harvey.	D. T. Lewis.
C. Titley.	R. J. Cook.	S. R. Thomas.
H. J. Jones.		

November.

T. H. Miller.	R. W. Murray.	R. Cuff.
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Apothecaries' Society.

Final Examination for the Licence.

J. M. Prendergast.	Z. Prentice.	W. J. Coles.
J. H. Dunlop.	J. B. Trapp.	J. C. Underwood.
P. Pigott.	H. L. Manby.	H. L. Cortis.
T. R. Atkinson.	W. H. Hart.	E. O. Stuart.
E. S. Dashwood.	B. Scott.	A. Edensor.
T. B. Luscombe.	J. Rees.	J. H. Greenway.
E. S. Cockell.	W. T. Harris.	

First Examination for the Licence.

A. Edensor.	D. T. Key.	H. L. Cortis.
J. Davies.	R. P. Samut.	J. C. Underwood.
A. L. Tireman.	W. H. Hart.	J. J. Faraker.
J. B. Berry.	E. T. Trevor.	J. H. Greenway.
W. S. Stables.	B. Scott.	G. H. Graham.

*University of London.**Matriculation Examination.**January.*

L. Bidwell.	F. B. W. Phillips.
H. E. Craig.	S. E. Prall.
W. N. Ridsen.	

June.

F. F. Burghard.	W. P. Le Feuvre
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*MEDALLISTS AND PRIZEMEN, 1880-81.**JULY, 1881.**The Treasurer's Gold Medal for Surgery.*

Lockhart Edward Walker Stephens, Emsworth.

Beane Prize for Pathology.

Edwin A. Starling, St. Leonards-on-Sea.

Michael Harris Prize.

Albert Martin, Wellington, New Zealand.

Third Year's Students.

Thomas Carr, Brixton, First Prize, £35.

William Thos. Frederick Davies, Swansea, Second Prize, £20.

Walter Thomas Harris, Ipplepen, Devon, Certificate.

John Oscroft Littlewood, Ashfield, Certificate.

John Henry Booth, Chesterfield, Certificate.

Second Year's Students.

Albert Martin, Wellington, New Zealand, First Prize, £25.

Arthur Ernest Larking, London, Second Prize, £10.

Francis Heatherley, London, Certificate.

John Herbert Hawkins Manley, West Bromwich, Certificate.

Thomas Hugh Miller, Virginia, Certificate.

Allan Glaisyer Minns, Thetford, Norfolk, Certificate.

First Year's Students.

Geo. Elliott Caldwell Anderson, Oudtsboorn, Cape Colony, First Prize, £50.

Reginald Maurice H. Randell, Sydenham, Second Prize, £25.

Alfred Herbert Tubby, Kennington, Certificate.

Ernest Willmer Phillips, Brighton, Certificate.

William Henry Bowes, Herne Bay, Certificate.

*SEPTEMBER, 1881.**Open Scholarship in Arts.*

Albert Edward White.

Open Scholarship in Science.

John Wychenford Washbourn, Gloucester.

Michael Harris Prize, 1882.

George Elliott Caldwell Anderson, Oudtsboorn, Cape Colony.

Pupils' Physical Society.

Session, 1881-82.

Honorary President.—Dr. WILKS.

Presidents.

Messrs. E. L. Adeney, M.B., E. H. Booth, H. Blatherwick, J. I. Boswell, W. C. Dendy, W. Fowler, B.Sc., T. B. Luscombe, R. A. Milligan, W. H. C. Newnham, M.B., G. N. Pitt, J. A. P. Price, M.B., W. W. Pryn, B. N. Rake, M.D., R. J. Ryle, B. Scott, F. H. Shaw, E. A. Starling, L. E. W. Stephens, J. B. Trapp, P. Warner, G. J. Wilson.

Honorary Secretaries.—C. J. SYMONDS, M.S.; R. E. CARRINGTON, M.D.

PRIZEMEN FOR THE SESSION, 1880-81.

To Mr. F. H. Shaw, £10, for his Paper on "A Case of Intestinal Obstruction."

To Mr. R. A. Milligan, £5, for his Paper on "Excision of Joints."

To Mr. B. Scott, £2 10s., for his Paper on "Ulcerative Endocarditis."

To Mr. J. B. Trapp, £2 10s., for his Paper on "Urethral Stricture."

To Mr. T. B. Luscombe, £5, as the Member who had distinguished himself most in the Debates of the Session.

CLINICAL APPOINTMENTS HELD IN THE YEAR 1881.

RESIDENT HOUSE PHYSICIANS.

W. A. Lane, M.B., B.S.	E. Penny, M.B.
B. N. Rake, M.D.	J. Mackern, M.D.
J. W. Meek, M.D.	G. J. Wilson.
P. Warner.	

RESIDENT HOUSE SURGEONS.

J. S. Crook.	P. M. Wood.
J. W. Sanders.	A. Scott.
J. Coek.	W. T. Crew.

RESIDENT OBSTETRIC ASSISTANTS.

John Smith, M.B.	G. R. Marsh, M.B.	P. M. Wood.
H. T. Bassett, M.B.	W. E. Fielden.	L. Burroughs.
W. W. Pryn.	D. C. Trott.	P. Warner.
E. A. Starling.	W. H. C. Newnham, M.B.	R. A. Milligan.

SURGEONS' DRESSERS.

P. Warner.	L. Stokes.	W. C. Dendy.
R. A. Milligan.	L. E. Shaw, M.B.	H. Blatherwick.
C. S. Harper.	G. S. Pollard.	J. E. Viney.
E. L. Adeney, M.B.	B. Scott.	T. Unicume.
J. W. Nicholson.	E. H. Booth.	F. Eastes.
T. M. Day.	R. H. Perks.	J. H. Lister.
L. Burroughs.	J. B. Trapp.	J. A. P. Price, M.B.
W. H. W. Strachan.	J. W. Hodgson.	L. E. W. Stephens.

CLINICAL ASSISTANTS.

J. I. Boswell.	L. Stokes.	J. J. Udale.
J. F. Spong.	G. F. Dixon.	J. Rigby.
W. C. Hearnden.	F. H. Shaw.	J. M. Owen.
H. Hawksworth.	H. G. Ashwell.	C. S. Harper.
W. H. C. Newnham.	H. A. Fotherby.	O. J. Currie, M.B.
G. T. Woolley.	L. E. Shaw, M.B.	W. R. Dakin, M.B.

DRESSERS IN THE EYE WARDS.

D. C. Trott.	W. A. Lane, M.B., B.S.	H. A. Clowes.
F. H. Shaw.	P. H. Gardner.	W. E. Fielden.
T. B. Luscombe.	O. J. Currie, M.B.	E. L. Adeney, M.B.
T. Unicume.	R. A. Milligan.	J. I. Boswell.
H. T. Bassett, M.B.	T. M. Day.	H. E. Richardson.
W. T. Crew.	J. C. Pincott.	J. M. Owen.

DENTAL SURGEONS' DRESSERS.

J. V. Salvage.	H. H. Wright.	E. Elliott.
W. E. Audland.	H. A. S. Mahomed.	W. L. W. Marshall.
E. S. Tresidder.	J. B. Berry.	G. P. Longman.
A. E. C. Woodhouse.	H. P. Keatinge.	H. A. B. Davies.

MEDICAL WARD CLERKS.

J. H. Kinch.	E. H. Booth.	W. J. C. Tomalin
G. R. Green.	F. C. Payne.	C. E. Beebe.
J. C. Underwood.	J. H. Cuolahan.	R. J. Ryle.
L. E. W. Stephens.	W. D. J. Morris.	J. H. Booth.
E. S. Dashwood.	H. T. Sells.	J. O. Littlewood.
R. H. Perks.	E. T. Trevor.	D. T. Key.
A. G. Wildey.	H. E. Richardson.	J. J. Tomney.
J. B. Howell.	W. C. Dendy.	H. B. Todd.
J. A. P. Price.	B. P. Bartlett.	E. Wakelam.
H. E. Rowell.	J. B. Trapp.	E. Apthorp.
H. Blatherwick.	H. P. Berry.	W. Growse.
F. E. Hubbard.	T. Cardwell.	W. T. Harris.
J. H. Champ.	F. Eastes.	H. G. Plimmer.
W. R. Dakin.	J. H. Greenway.	T. Carr.
W. S. N. Shorthouse.	A. De Winton.	W. R. Etches.
J. W. Hodgson.	G. N. Pitt.	W. Wilson.
J. G. Milnes.	J. H. Gibson.	W. E. Audland.
J. E. Viney.	J. M. Griffin.	L. McE. Anderson
G. P. Longman.	W. E. Rudd.	J. F. Saunders.
J. J. Faraker.	E. W. Roberts.	R. P. Samut.
J. H. Lister.	D. T. Edmunds.	E. G. Hunt.
W. H. Hart.		

ASSISTANT SURGEONS' DRESSERS.

G. P. Longman.	W. E. Audland.	W. Wilson.
H. W. Moor.	E. S. Dashwood.	H. G. Plimmer.
F. N. Shillingford.	E. R. S. Lipscomb.	C. E. Beebe.
J. E. Viney.	R. J. Ryle.	J. H. Cox.
W. Growse.	D. T. Edmunds.	T. F. B. Palmer.
E. T. Trevor.	J. A. P. Price.	W. Howard.
H. C. Dixon.	J. H. Gibson.	E. Roberts.
B. P. Bartlett.	H. A. B. Davies.	J. B. Berry.
E. H. Booth.	W. R. Dakin.	R. Browne.
H. T. Sells.	H. E. Richardson.	J. J. Faraker.
W. C. Dendy.	D. T. Key.	A. P. H. Griffiths.
E. W. Roberts.	W. T. Harris.	J. J. Prendergast.
T. F. W. Rowlands.	G. H. Graham.	M. O'Kane.
J. H. Lister.	L. Powell.	H. R. Mead.
W. H. Hart.	W. B. Skelton.	L. A. Dunn.
A. De Winton.	W. R. Etches.	E. S. Tresidder.
G. Utting.	R. P. Samut.	H. W. Pigeon.
T. Cardwell.	T. Carr.	H. P. Berry.
J. M. Griffin.	A. E. C. Woodhouse.	W. J. C. Tomalin.
W. E. Rudd.	W. H. Moore.	G. Kendall.
J. H. Greenway.	S. Worthington.	H. H. Wright.
J. H. Booth.	H. B. Todd.	A. J. Dalton.
J. O. Littlewood.	L. McE. Anderson.	

DRESSERS IN THE SURGERY.

T. Cardwell.	S. B. A. Edsall.	J. J. Prendergast.
M. O'Kane.	J. A. Marsden.	E. E. Masters.
J. M. Griffin.	L. McE. Anderson.	W. Watson.
J. H. Greenway.	G. W. B. Slader.	G. A. Johnson.
H. E. Richardson.	L. A. Dunn.	A. E. Larking.
H. H. Wright.	W. T. F. Davies.	H. W. Pigeon.
W. H. Moore.	S. Worthington.	D. T. Lewis.
T. Carr.	A. L. Scott.	L. J. Kidd.
H. G. Plimmer.	W. L. W. Marshall.	J. P. Martin.
G. Kendall.	C. E. Beebe.	W. D. Smallpeice.
E. R. S. Lipscomb.	W. Wilson.	A. J. Carter.
A. E. C. Woodhouse.	W. C. Spiller.	F. W. H. Penfold.
E. S. Tresidder.	H. G. Hilbers.	H. P. Keatinge.
J. F. Saunders.	J. E. Viney.	C. H. L. Meyer.
J. H. Cox.	J. J. Faraker.	A. M. Sutton.
A. Searle.	W. Howard.	A. T. F. Browne.
J. O. Littlewood.	E. Wakelam.	J. C. Bates.
R. H. Browne.	G. C. Stamper.	C. S. Spong.
D. T. Key.	A. P. H. Griffiths.	A. L. Tireman.
T. F. B. Palmer.	F. Pearse.	T. B. Winter.
F. E. Cave.	M. A. Muirhead.	C. Y. Shuter.
H. R. Mead.	A. L. Paliologus.	W. Hind.
H. H. W. Button.	R. A. Baillie.	M. Parry-Jones.
L. Powell.	E. O. Newland.	E. Linnell.

AURAL SURGEON'S DRESSERS.

H. E. Richardson.	H. B. Todd.	W. D. J. Morris.
J. G. Milnes.	H. A. B. Davies.	E. Elliott.
A. S. Topham.	F. Eastes.	W. L. W. Marshall.
H. R. Osborne.	T. Carr.	G. P. Longman.
A. H. Jackson.	J. B. Howell.	

ASSISTANT PHYSICIANS' CLERKS.

F. V. Duckworth.	J. B. Howell.	H. T. Sells.
J. A. Fraser.	J. H. Champ.	W. H. Hart.
C. R. O. Garrard.	H. C. Dixon.	E. T. Trevor.
R. Bredin.	L. E. W. Stephens.	P. H. Gardner.
J. Dowson.	H. Blatherwick.	E. W. Roberts.
F. N. Pedley.	G. P. Longman.	J. H. Greenway.
F. E. Row.	W. D. J. Morris.	D. T. Edmunds.
E. O. Stuart.	R. Bredin.	H. C. Ensor.
J. C. Underwood.	J. C. Underwood.	R. J. Ryle.
J. W. Hodgson.	O. J. Currie.	J. H. Gibson.
Z. Prentice.	W. R. Dakin.	

POST-MORTEM CLERKS.

E. G. Hunt.	A. G. Wildey.	J. V. Salvage.
D. T. Edmunds.	A. De Winton.	H. H. Du Boulay.
J. H. H. Williams.	J. B. Berry.	M. A. Muirhead.
W. J. C. Tomalin.	B. N. Rake.	A. L. Scott.
H. B. Todd.	Q. R. Veitch.	H. T. Sells.
J. A. Fraser.	E. T. Trevor.	L. Powell.
J. J. Prendergast.	E. S. Tresidder.	J. O. Littlewood.
E. D. Minter.		

OBSTETRIC OUT-PATIENTS CLERKS.

A. Bolton.	R. Bredin.	J. J. Prendergast.
P. Pigott.	J. W. Hodgson.	R. A. Milligan.
T. Unicume.	F. N. Shillingford.	Q. R. Veitch.
G. J. Currah.	F. E. Hubbard.	F. Eastes.
W. D. J. Morris.	L. E. W. Stephens.	G. P. Longman.
H. Hawksworth.	J. A. P. Price.	H. C. Ensor.
T. F. W. Rowlands.	W. E. Audland.	W. L. W. Marshall.
R. H. Perks.	W. H. Hart.	A. G. Minns.

OBSTETRIC WARD CLERKS.

A. P. H. Griffiths.	G. Utting.	J. H. H. Williams.
W. Growse.	J. H. Cuolahan.	J. H. Champ.
J. H. Cox.	H. H. Wright.	J. A. Marsden.
W. T. Harris.		

EXTERN OBSTETRIC ATTENDANTS.

J. H. Cox.	T. Carr.	B. P. Bartlett.
A. S. Topham.	D. T. Key.	J. M. Griffin.
J. H. Cuolahan.	E. W. Roberts.	J. P. Martin.
W. H. Moore.	R. H. Browne.	H. Davenport.
E. Apthorp.	H. B. Todd.	G. N. Pitt.
M. A. Muirhead.	W. C. Dendy.	M. Carnelley.
W. Howard.	E. H. Booth.	S. Worthington.
T. Cardwell.	W. H. Hart.	C. Woodhouse.
G. H. Graham.	J. A. P. Price.	H. Howard.
J. E. Viney.	J. H. Lister.	A. De Winton.
H. E. Rowell.	G. P. Longman.	F. Pearse.
W. S. N. Shorthouse.	E. D. Minter.	W. L. W. Marshall.
J. C. Underwood.	H. T. Sells.	E. J. Wenyon.
J. Dowson.	J. G. Milnes.	S. B. A. Edsall.
A. G. Wildey.	T. F. B. Palmer.	W. E. Rudd.
F. Eastes.	G. Kendall.	H. P. Berry.
W. Wilson.	H. W. Pigeon.	R. P. Samut.
J. F. Saunders.	J. A. Marsden.	L. McE. Anderson.
S. O. Stuart.	E. Linnell.	W. E. Audland.
L. E. W. Stephens.	W. P. West.	

SURGICAL WARD CLERKS.

J. G. Milnes.	A. S. Topham.	W. H. Brenton.
L. Powell.	G. Kendall.	A. G. Minns.
T. F. B. Palmer.	A. L. Paliologus.	T. H. Miller.
J. H. Gibson.	F. Pearse.	R. L. Knaggs.
H. A. B. Davies.	W. H. Moore.	E. D. Minter.
W. D. Smallpeice.	H. G. Plimmer.	H. C. Ensor.
E. Roberts.	G. W. B. Slader.	R. W. Brogden.
W. T. Harris.	A. P. H. Griffiths.	W. W. Floyer.
R. J. Ryle.	M. A. Muirhead.	W. A. Aikin.
S. B. A. Edsall.	E. S. Tresidder.	E. E. Masters.
L. McE. Anderson.	H. W. Pigeon.	W. Fowler.
W. Wilson.	H. R. Mead.	A. Martin.
S. Worthington.	T. B. Jacobson.	W. Watson.
L. A. Dunn.	J. P. Martin.	J. H. H. Manley.
W. C. Spiller.	A. L. Scott.	G. H. Johnson.
W. T. F. Davies.	St. J. O. Rands.	R. T. Westbrook.
W. R. Etches.	J. C. Bates.	W. T. Hodge.
B. N. Moorhouse.	W. L. W. Marshall.	G. F. Hugill.
J. J. Prendergast.	A. Linnell.	C. E. Bean.
J. A. Marsden.	A. T. F. Brown.	A. H. Dodd.
M. O'Kane.	T. B. Winter.	H. E. Counsell.
H. P. Berry.	A. L. Tireman.	S. R. Thomas.
W. Howard.	A. W. Clark.	F. B. W. Phillips.
E. Wakelam.	A. C. Deare.	J. I. Parsons.
W. J. C. Tomalin.	C. D. Muspratt.	

ASSISTANT SURGEONS' CLERKS.

W. B. Colquhoun.	W. R. Etches.	S. B. A. Edsall.
R. A. Bindley.	W. Fowler.	C. W. Crealock.
M. A. Muirhead.	W. L. Braddon.	J. A. Bradbury.
J. A. Marsden.	H. F. Knyvett.	W. H. Dodd.
A. E. Larking.	J. B. Berry.	F. C. Butt.
J. H. Greenway.	C. F. Wakefield.	A. Sutton.
F. W. Foster.	A. J. Dalton.	T. John.
C. N. Graham.	C. Pink.	A. E. Taylor.
E. W. Phillips.	A. H. Tubby.	C. Fryer.
A. E. C. Woodhouse.	H. A. Reed.	H. P. Keatinge.
J. D. Howe.	S. H. Mangin.	

GUY'S HOSPITAL.

MEDICAL AND SURGICAL STAFF.

1883.

Consulting Physicians.

SIR WILLIAM GULL, Bart., M.D., D.C.L., F.R.S.; G. OWEN REES, M.D., F.R.S.

Physicians.

S. WILKS, M.D., F.R.S.; F. W. PAVY, M.D., F.R.S.; W. MOXON, M.D.;
C. HILTON FAGGE, M.D.

Assistant Physicians.

P. H. PYE-SMITH, M.D.; FREDERICK TAYLOR, M.D.; J. F. GOODHART, M.D.;
F. A. MAHOMED, M.D.

Consulting Surgeons.

E. COCK, Esq.; J. BIRKETT, Esq.

Surgeons.

THOMAS BRYANT, Esq.; A. E. DURHAM, Esq.; H. G. HOWSE, M.S.;
N. DAVIES-COLLEY, M.C.

Assistant Surgeons.

R. CLEMENT LUCAS, B.S.; C. H. GOLDING-BIRD, M.B.; W. H. A. JACOBSON, Esq.
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Dean.—Dr. F. TAYLOR.

WINTER COURSES.

The Winter Session commences October 1st and ends March 31st.

LECTURES.

Medicine.—Dr. PAVY and Dr. MOXON.

Mondays, Wednesdays, and Fridays, at Three.

Clinical Medicine.—Dr. WILKS, Dr. PAVY, Dr. MOXON, and Dr. FAGGE.

Saturdays, at Half-past One.

Surgery.—Mr. BRYANT and Mr. DURHAM.

Tuesdays and Thursdays, at Half-past Three, and Saturdays, at a Quarter to Three.

Clinical Surgery.—Mr. BRYANT, Mr. DURHAM, Mr. HOWSE, and Mr. DAVIES-COLLEY.

Wednesdays, at Half-past One.

Ophthalmic Surgery.—Mr. HIGGENS.

Thursdays, at Three.

Anatomy, Descriptive and Surgical.—Mr. HOWSE and Mr. DAVIES-COLLEY.

Tuesdays, Wednesdays, Thursdays, and Fridays, at Nine.

Physiology and General Anatomy.—Dr. PYE-SMITH.

Mondays, Wednesdays, and Fridays, at a Quarter-past Four.

Clinical Lectures on Midwifery and Diseases of Women.—Dr. GALABIN.

Wednesdays, at Half-past One.

Chemistry.—Dr. DEBUS and Dr. STEVENSON.

Tuesdays, Thursdays, and Saturdays, at Eleven.

Experimental Physics.—Prof. A. W. REINOLD, F.R.S.

Mondays and Wednesdays, at Eleven.

Comparative Anatomy and Zoology.—Dr. BRAILEY.

Mondays and Thursdays, at a Quarter to Two.

DEMONSTRATIONS.

Practical Surgery.—Mr. LUCAS.

Surgical Classes.—Mr. JACOBSON, *Daily.*

Practical Anatomy.—Dr. W. H. WHITE, Mr. W. A. LANE, and

Mr. J. A. P. PRICE, *Demonstrators.*

And two Assistant Demonstrators.

Morbid Anatomy.—Dr. GOODHAET and Dr. MAHOMED.

Daily, at Half-past Two.

Cutaneous Diseases.—Dr. PYE-SMITH.

Tuesdays, at Twelve.

Practical Physiology.—Mr. GOLDING-BIRD.

Tuesdays, at Half-past One, Wednesdays and Saturdays, at One.

Morbid Histology.—Mr. SYMONDS.

Three days in the week.

Practical Pharmacy.

SUMMER COURSES.

The Summer Session begins May 1st and ends July 31st.

LECTURES.

Materia Medica and Therapeutics.—Dr. TAYLOR.

Mondays, Tuesdays, and Thursdays, at Two.

Midwifery and Diseases of Women.—Dr. GALABIN

Tuesdays, Wednesdays, Thursdays, and Fridays, at Nine.

Medical Jurisprudence.—Dr. STEVENSON.

Tuesdays, Thursdays, and Saturdays, at Ten.

Clinical Medicine.—Dr. PYE-SMITH, Dr. F. TAYLOR, Dr. GOODHART, and Dr. MAHOMED.

Wednesdays, at Half-past One.

Clinical Surgery.—Mr. LUCAS, Mr. GOLDING-BIRD, Mr. JACOBSON, and Mr. SYMONDS.

Fridays, at Half-past One.

Clinical Lectures on Diseases of Women.—Dr. GALABIN.

Tuesdays, at Half-past One.

Pathology.—Dr. FAGGE, *Saturdays, at Nine.*

Hygiene.—Mr. GEORGE TURNER, *Mondays and Fridays, at Twelve.*

Mental Diseases.—Dr. SAVAGE.

Tuesdays, at Eleven, and Fridays, at Half-past Ten.

Botany.—Mr. BETTANY.

Tuesdays, Thursdays, and Saturdays, at a Quarter past Eleven.

Dental Surgery.—Mr. MOON.

DEMONSTRATIONS.

Practical Chemistry.—Mr. C. E. GROVES, F.R.S.

Mondays, Wednesdays, and Fridays, Ten to One.

Operative Surgery.—Mr. LUCAS.

Mondays, Wednesdays, and Fridays, at Four.

Morbid Anatomy.

Cutaneous Diseases.

Morbid Histology.

Practical Pharmacy.

Surgical Classes.

With the same arrangements as during the Winter Session.

Practical Courses and University Classes in Anatomy, Physiology, Botany, Comparative Anatomy, and Natural Philosophy.

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ASTLEY COOPER PRIZE.

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And it is expressly declared in the Will “That no Physician or Surgeon, or other officer for the time being, of Guy’s Hospital or of St. Thomas’s Hospital, in the Borough of Southwark, nor any person related by blood or affinity to any such Physician or Surgeon, for the time being, or to any other Officer for the time being in either of the said Hospitals, shall at any time receive or be entitled to claim the Prize.” But, with the exception here referred to, this Prize is open for competition to the whole world.*

Candidates are informed that their Essays, either written in the English Language, or, if in a Foreign Language, accompanied by an English translation, must be sent to Guy’s Hospital on or before January 1st, 1886, addressed to the Physicians and Surgeons of Guy’s Hospital.

Each Essay or Treatise must be distinguished by a Motto, and be accompanied by a sealed envelope containing the name and address of the Writer. None of the envelopes will be opened except that which accompanies the successful Treatise. The unsuccessful Essays or Treatises, with the illustrative preparations or drawings, will remain at the Museum of Guy’s Hospital until claimed by the respective writers or their agents.

* The Prize cannot be awarded to any Essay that is the joint production of two or more authors.

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